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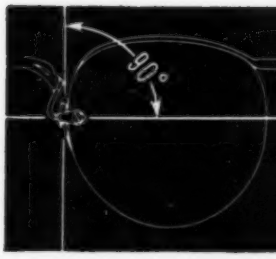

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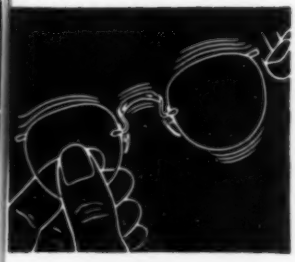

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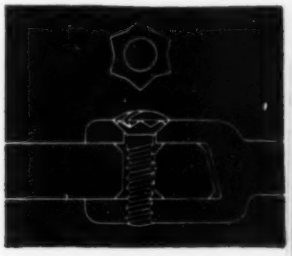

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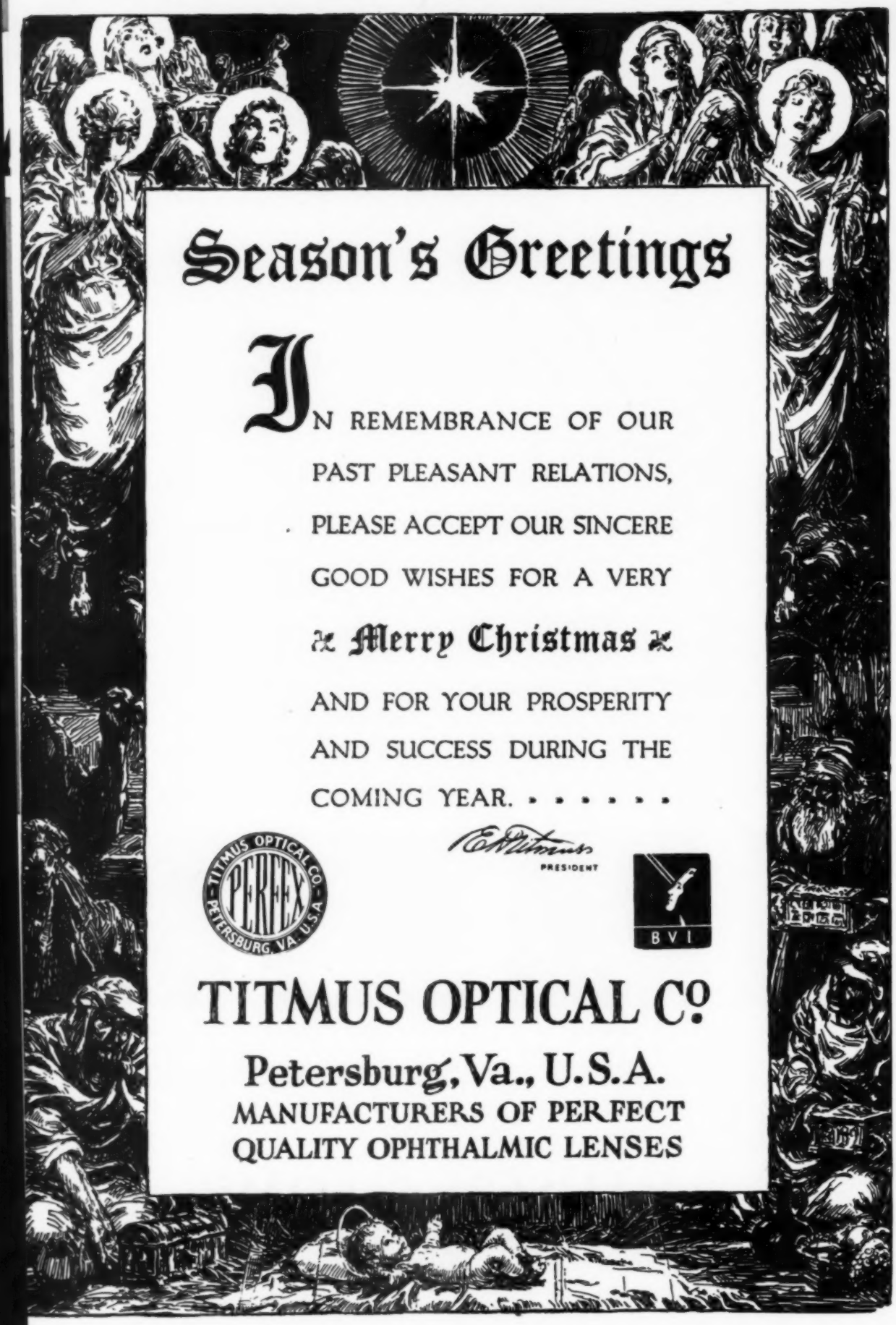

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Santa
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(with apologies to Clement Clarke Moore*)*

'Twas the night before Christmas
and all Santa's strength
Had been sapped by the battle
with blur at arm's length.

So when to my wondering eyes
did appear
The miniature sleigh
and the tiny reindeer,
The little old driver
—St. Nick, by his clothes—
Was scanning a road map . . .
a foot from his nose!

"Now, Prancer! Now, Dasher!
Now, Comet and Cupid!
It must be these glasses—
I can't be this stupid!"

As up to the house-top the coursers they soared
St. Nick missed a detour—"Wrong chimney!" he roared.
Too late! As I peered in the hearth came a shout
And from down in the cellar a faint "Let me out!"

He had a broad face
and a round little belly
That shook when he laughed—
but his vision was smelly.
Though the stockings were hung
by the chimney with care,
Old Nick couldn't see
to tell which gift went where!

Then he sprang at his sleigh
with a cross "Giddyap!"
Turning southward to reach
the North Pole on the map.
And I heard him exclaim
as he drove out of sight,
"I must see my doctor—
sumthin' ain't right!"

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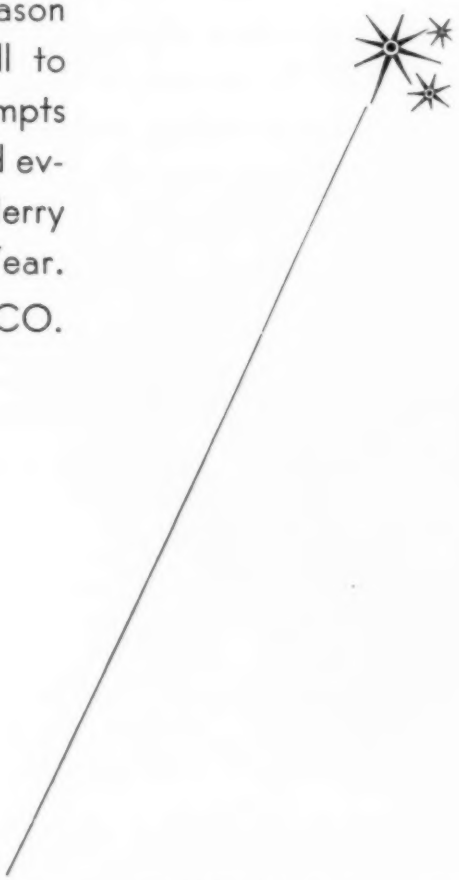

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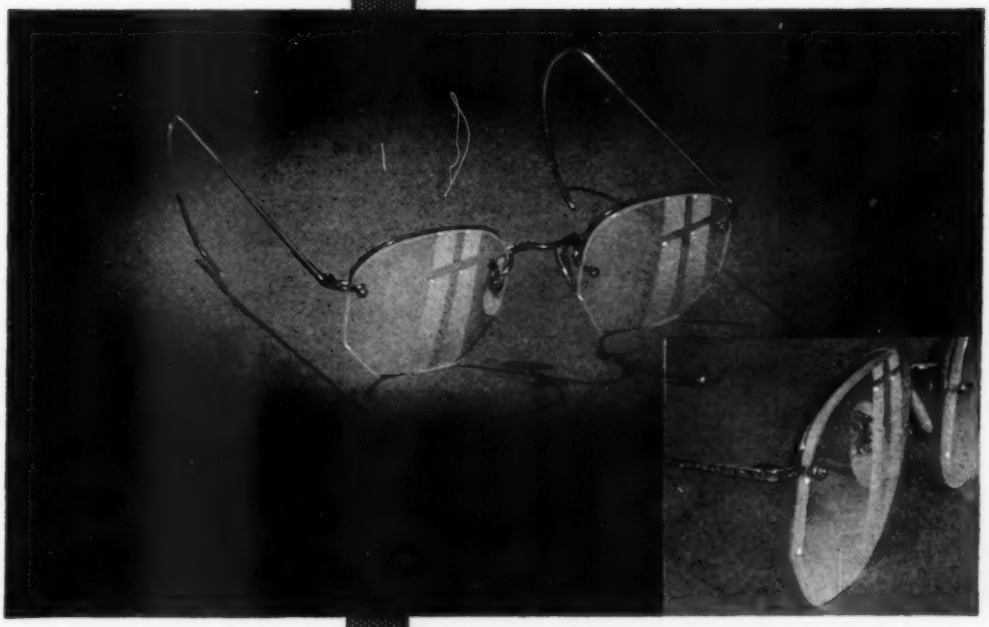

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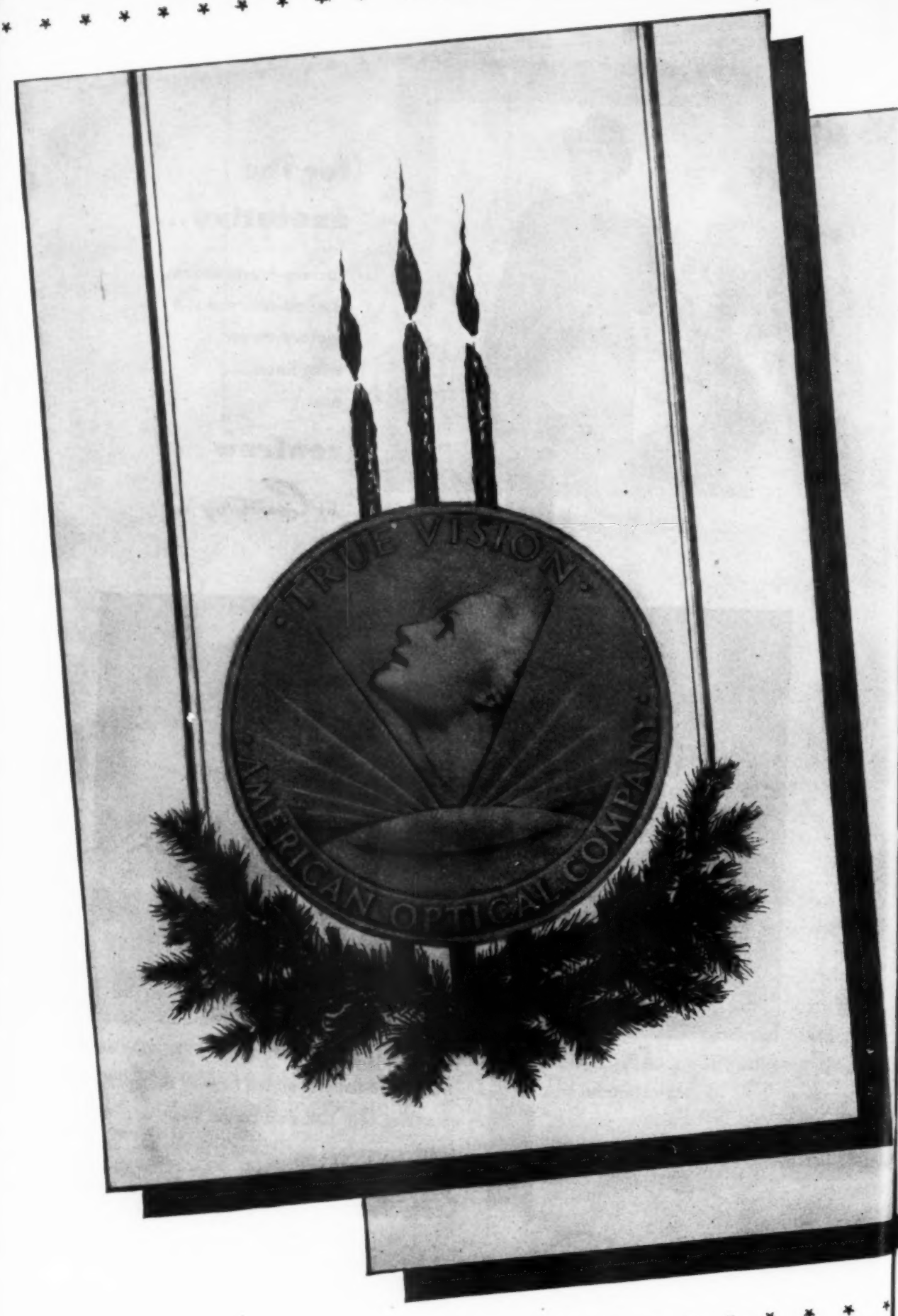
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for a New Year of Health and Happiness

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Vol. 24 DECEMBER, 1947 No. 12

TWO IMPORTANT DIAGNOSTIC POINTS IN TROPIAS*

J. Donald Kratz†
Souderton, Pennsylvania

In discussing tropias, first consideration must be given to classification and thus a differential diagnosis of the various types. This is necessary because of the great variance in prognosis depending on the type of tropia which may be encountered. For today's considerations, I feel the most useful classification is one based on etiology. With this type of classification we can describe five distinctly different types of tropia, varying in prognosis and in etiology.

The first of these is the anatomical tropia. The anatomical tropia in its pure form will have as its etiology some anatomical defect, either congenital or acquired (through surgery or accident). Anatomically we may have a malformation of some of the orbital structure or perhaps the failure of development of some of the extra-ocular muscles or nerves. The congenital anatomical tropia is frequently associated with some other defect of development such as a cleft palate, coloboma of the choroid or many others which could be mentioned. The anatomical tropia as a result of injury could be distinguished by the history where the patient would report injury or operation. In this respect it might be mentioned that a tropia developing after operation (such as a tonotomy) for the correction of a previously existing tropia, will have a poor prognosis because it is then essentially an anatomical tropia and the anatomical type tropias are generally not amenable to orthoptic training since missing structures cannot be realigned.

In etiological classification the second type to consider is the paradoxical motor lesion. This tropia is characterized by a completely erroneous behavior which follows no routine pattern but which varies haphazardly from time to time with no comprehensive explanation. These tropias may be encountered in hysterical individuals or as a result of shock or even with no other apparent symptoms. One eye may devi-

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*Read before the annual meeting of the American Academy of Optometry, Philadelphia, Pennsylvania, May 20, 1947. For publication in the December, 1947, issue of the AMERICAN JOURNAL OF OPTOMETRY AND ARCHIVES OF AMERICAN ACADEMY OF OPTOMETRY.

†Optometrist. Fellow, American Academy of Optometry. Assistant Professor of Optometry, Pennsylvania State College of Optometry.

ate outwards while the other undergoes levoversion and the next trial may elicit a complete reversal of these characteristics. This type tropia is encountered very infrequently and is due possibly to the failure of certain nerve centers to develop properly. A tropia of this character may be summed up by saying a normal stimulus will result in a completely incongruous response.

The third classification in this system is that of an accommodative tropia. Little need be said concerning this type of tropia since most optometrists will be quick to recognize its characteristics such as high hypermetropic ametropia binocularly and a convergent tropia which will be relieved by application of the proper correcting lenses. The etiology of this type is the relationship between convergence and accommodation. The accommodative action necessary for clear vision is quite high (enough to overcome the ametropia) and this effort stimulates convergence by reflex innervation. If the stimulus is sufficiently strong, fusion will be broken and the resultant convergent tropia will appear. Correction of the ametropia will relieve the necessity for accommodative action and the circle will be broken thus allowing fusion to again maintain the correct ocular postures for single binocular vision. The prognosis of these tropias is good, and ultimately the eyes will assume a straight ahead position when wearing the correction without the need for additional orthoptics.

The fourth and fifth groups are the important ones from the standpoint of clinical practice because of their many forms and varying prognosis. Let us call the fourth group the tonic tropia. The classical thoughts on this type are concerned with the tonus of the extra-ocular muscles and the innervational conditions which set up this tonus. The thought is that if one of a pair of yoked muscles has a normal innervational stimulus and the other a hypertonic innervational pattern the result will be a tropia. No doubt this has a kernel of truth, but the orthoptic significance of this statement is frequently overlooked or not clearly adduced. If this is the situation, then simple calesthatic exercises will not remedy the situation unless by accident, since every movement in one direction must have a movement in the opposite direction following it. If the eyes undergo levoversion, they must then undergo dextroversion. The only remedy of this situation which directly attacks the problem is the building of fusional amplitude. Once the fusional amplitude is sufficient to overcome the tonic disability, the tropia is clinically corrected; but in these cases if fusion is broken artificially or otherwise, the eyes will usually deviate to their former position of tropia.

It is this relationship which prevails in phorias of any degree. In orthoptic training with this type tropia we do not then correct the tropia *per se* but actually build a defense against it. The prognosis of this group is variable and depends on factors such as age of patient, angle of tropia, age of onset, depth of amblyopia, degree of suppression, and angle of anomaly.

The last group in this system of classification is the paralytic tropia. The nature of a paralytic tropia is easily described by saying there is a paresis or paralysis of one or more of the extra-ocular muscles. In actual practice, however, it is sometimes difficult to determine the presence or absence of this type tropia as well as which muscle or muscles is the affected one. When the affected muscles are the internal or external recti, the problem is not difficult; but when the obliques or the superior or inferior recti are involved, the difficulties mount.

Diagnostically the most important clue to the presence of a paralytic tropia is the presence of diplopia in certain positions of the eye and its absence in others. Since the angle of the tropia increases as we move toward the affected muscle, we have a useful characteristic in diagnosis. Diplopia will occur in the direction of the affected muscle, and the image belonging to the affected eye will always precede the image of the normal eye. With this in mind it is possible to draw a diagnostic procedure whereby we can determine the afflicted muscle with certainty by means of the motor fields.

As a prelude to this discussion it is best to review briefly the pertinent points concerning the anatomy of the orbit. For our purposes we can consider the orbits as being triangular in shape with the inner walls parallel when looking down from above. The four recti muscles then have their origin at the apex of this triangle and proceed to the eyeball in a cone-shaped manner. The superior and inferior obliques have an effective origin at the inner walls of the orbits. With this anatomical picture it becomes clear that the effectivity of a specific muscle or muscle group varies with the position of the eye. See Figure 1.

If we examine the action of the extra-ocular muscles successively in the primary position, gaze to the right and gaze to the left, certain groups will have a paired action and are then called yoked muscles.

If the eyes are gazing to the right, the elevator muscles will be a right superior rectus and the left inferior oblique, and the depressors will be a right inferior rectus and the left superior oblique. On gazing to the left, a reversal of this situation will occur, the elevators being the left superior rectus and the right inferior oblique. The depressor

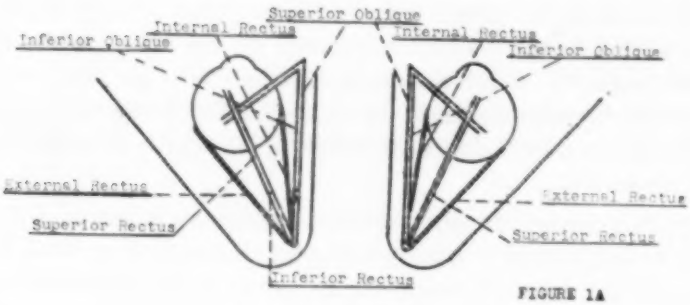


FIGURE 1A

Primary position of Eyes

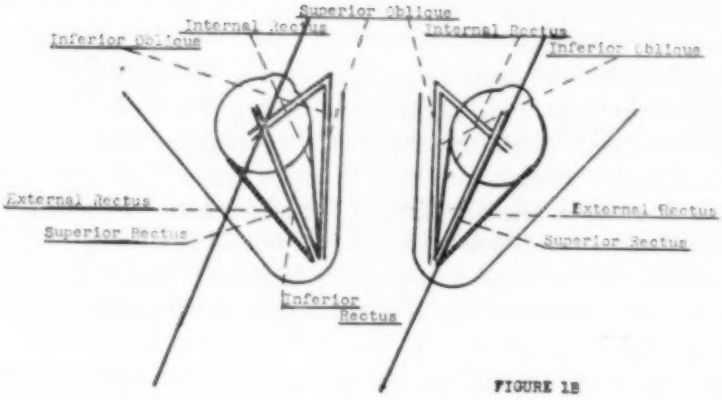
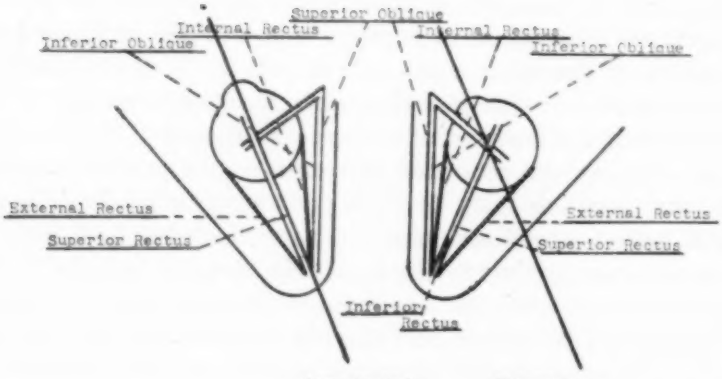


FIGURE 1B

Gaze to Right



Gaze to Left

FIGURE 1C

muscles in this situation will be the left inferior rectus and the right superior oblique. In the straight ahead position of the eyes (primary position) the action of the recti muscles and the oblique muscles are a mixture of rotation, elevation, and depression. Thus any movement of the visual axes from this position is a composite of the action of all of these muscles.

Since the action of the extra-ocular muscles varies with the position of the eye and the direction of the gaze, we can by a careful plotting of the motor fields determine which muscle or muscles is affected in a given situation. The simplest way of approaching this problem from a clinical standpoint is by construction of motor fields. A chart can be constructed having nine blocks of equal size corresponding to nine different fixation positions. The chart should be constructed to coincide with the actual position of the object as viewed by the patient. See Figure 2.

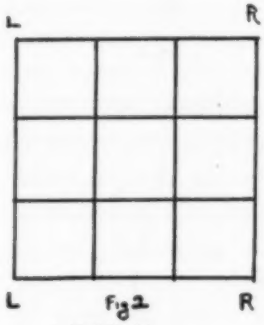


Fig 2

+ Red Light
O White Light.

(View from Patient)

In working with this chart the patient should have a red filter in front of the right eye and in most cases will not be wearing his correction. The patient thus will be seeing a red light with his right eye and the white light with his left eye when viewing a retinoscope bulb which can be used as a fixation object. This light is held approximately 20 inches from the patient and is moved in horizontal and vertical directions to coincide with the various fixation fields on the motor chart.

Taking paresis of the various extra-ocular muscles in sequence we can now determine the affected muscle with a relative degree of certainty.

PARESIS OF THE RIGHT EXTERNAL RECTUS

This muscle is the right hand rotator for the right eye. On directting the gaze to the right side the red light seen by the right eye will precede the white light which is fixated by the good left eye. The nor-

mal left eye will follow the white light but the right eye will be unable to do so resulting in an ever-widening separation between the white and the red image with the red image moving progressively to the right. Directing the gaze downward will result in a slight increase of the separation of the image because of the added convergence impulse which is not highly significant. Upward movement when gazing to the right will conversely have a tendency to decrease the separation due to a divergence impulse on directing the gaze upward, see Figure 3.

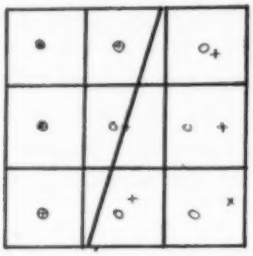


Fig 3

PARESIS OF THE LEFT INTERNAL RECTUS

Here the paresis is affecting the right hand rotator of the left eye. The effect will be noted when directing the gaze to the right, but the white light in this situation will precede the red with the gap widening as the gaze is progressively extended further to the right. The convergence factor will again operate upon directing the gaze upward or downward with fixation to the right but may be ignored as previously. See Figure 4.

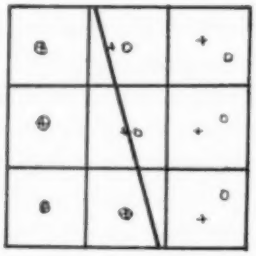


Fig 4

PARESIS OF THE LEFT EXTERNAL RECTUS

Here the motor field to the left will be impaired and diplopia will again occur, this time with fixation directed to the left. The white light will precede the red with an increasing separation as the gaze is

directed progressively to the left. Again superiorly the separation will be less than inferiorly. See Figure 5.

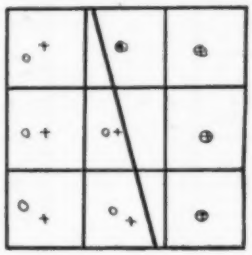


Fig 5

PARESIS OF THE RIGHT INTERNAL RECTUS

The paresis here will again become evident in the left hand motor field. The diplopia will increase on direction of the gaze to the left. The difference will be that in this situation the red light will precede the white with an ever greater separation. Directing the gaze upward and downward will again affect the separation of the lights, while looking to the left due to convergence influence. Separation here will be greater on directing the gaze upward. See Figure 6.

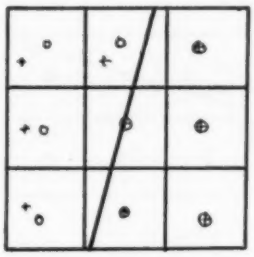


Fig 6

PARESIS OF THE RIGHT SUPERIOR RECTUS

The right superior rectus is the elevator for the right eye on right hand version. Therefore, the vertical diplopia field on the right hand side will show greater separation than the vertical diplopia field on the left hand side, where the right inferior oblique replaces the action of the right superior rectus. Since the right eye has the paresis the red light will precede the white with the movement in the direction of movement of the fixation object as diplopia occurs. See Figure 7.

PARESIS OF THE LEFT INFERIOR ORLIQUE

Paresis of the left inferior oblique will cause the same field characteristics as the right superior rectus except that since the left eye has the paresis the white light will be preceding the red. This characteris-

tic is true of the action in all yoked muscles. See Figure 8.

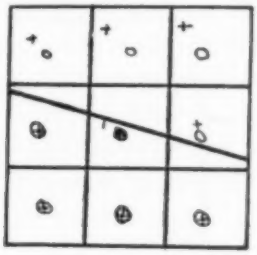


Fig 7

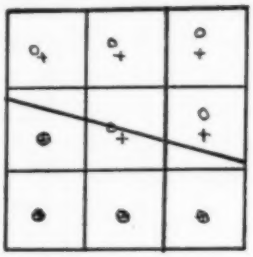


Fig 8

PARESIS OF THE LEFT SUPERIOR RECTUS

Here the superior vertical diplopia fields will be wider on the left hand side, since the left superior rectus has its main elevating action on levoversion. On dextroversion the left inferior oblique assumes the action of an elevator, therefore the effect of the paresis of the left superior rectus is at a minimum in this position. Since the left eye is paretic, the white light will be in precedence. See Figure 9.

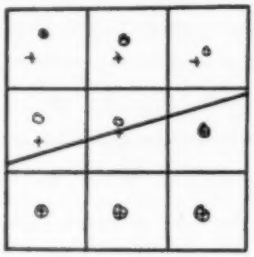


Fig 9

PARESIS OF THE RIGHT INFERIOR OBLIQUE

This being the yoke muscle with the left superior rectus the effect on the motor fields will be similar. On looking to the left you will have maximum diplopia. On looking to the right you will have minimum separation since in dextroversion the right superior rectus will become the elevator muscle. Here the red light will precede the white since the paralysis is on the right eye. See Figure 10.

PARESIS OF THE RIGHT INFERIOR RECTUS

This muscle being the depressor muscle in a position of dextroversion, we will have greatest vertical diplopia in the inferior field on the right hand side. With gaze directed to the left the diplopia will be minimized because the depressor action will be taken over by the right superior oblique. The diplopia will be characterized by the red light

preceding the white in the direction of movement of the fixation target since the paresis is on the right eye. See Figure 11.

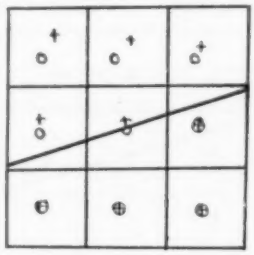


Fig 10

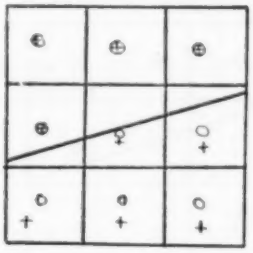


Fig 11

PARESIS OF THE LEFT SUPERIOR OBLIQUE

This muscle being the depressor for the left eye on right hand gaze the diplopia effect will be similar to the effect of the right inferior rectus with greater diplopia in the right inferior field and less on the left inferior field where the left inferior rectus will take over the depressor action. With paresis of the left superior oblique the white light will now precede the red light. See Figure 12.

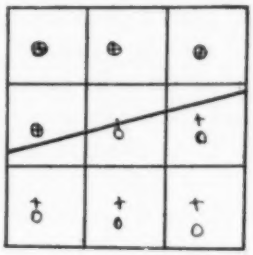


Fig 12

PARESIS OF THE LEFT INFERIOR RECTUS

Here the diplopia will be greatest in the inferior field on the left side, since the action of this muscle is chiefly centered in this direction. On fixation to the right the depressor action will be taken up by the left superior oblique and, therefore, the diplopia will be minimized. The paresis being on the left eye, the white light will precede the red. See Figure 13.

PARESIS OF THE RIGHT SUPERIOR OBLIQUE

This being the yoke muscle of the left inferior rectus, the motor fields will be similarly affected with greatest diplopia on the inferior field with gaze to the left. On dextro-version the depressor action of the right superior oblique will be replaced by the right inferior rectus and

diplopia will be at a minimum. Since the paresis is on the right eye, the red light will precede the white as the separation increases. See Figure 14.

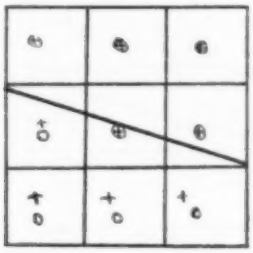


Fig 13

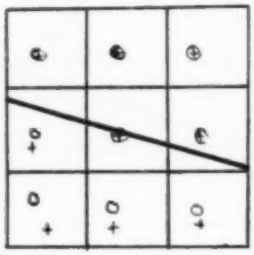


Fig 14

It can be seen from the discussion above that the action of these muscles is in a paired or yoked manner, and the action in the motor field is similar for the yoked muscles, except for the movement of the white and red lights. In all cases the light belonging to the eye having the paresis or paralysis will precede the light belonging to the normal eye. Pairing the muscles we see that the left inferior rectus and the right superior oblique were yoked muscles. The left superior oblique and right inferior rectus will also be paired in action. The right inferior oblique and the left superior rectus will have a similar or paired action. The left inferior oblique and the right superior rectus are still another pair of yoked muscles, with their effect in diplopia field centered in the vertical fields. The other two pairs of yoked muscles are the right internal rectus and the left external rectus, with the left internal rectus and the right external rectus being the last set of yoked muscles.

In spite of the analysis being given with respect to the linked nature of these yoked muscles, paralysis will not most typically occur in this manner. During this session of the Academy it has been shown that the innervation of the extra-ocular muscles is accomplished by three of the cranial nerves.[1] The third nerve innervates the internal rectus, superior rectus, inferior rectus, and inferior oblique muscles. The fourth cranial nerve innervates the superior oblique and the sixth or abducens innervates the external rectus. Since the third cranial nerve affects four of the extra-ocular muscles, a paresis or nuclear lesion of this nerve will have a very great effect on the motor field. Since the third nerve also indirectly innervates the ciliary muscle and iris of the eye, effects on these structures may also be apparent. In determining whether it is the

[1]Gilda Coppola. The Extra-Ocular Muscles—Their Functional Requirements and Nervous Control. Am. J. Optom. and Arch. Am. Acad. Optom. 24.9, 443-450, 1947.

right or left third nerve paralysis we only need to find which light is in precedence in the various motor fields. If the red light is in precedence, it will be the right side and if the white light is in precedence it will be the left side.

According to most authorities, paralysis of the external rectus which is innervated by the abducen's nerve is the paralysis encountered most frequently. According to Bielchowsky, 60 per cent of the paralytic tropias are of this type. The explanation of the frequency of this condition lies most probably in the anatomy of the abducen's nerve. The second longest cranial nerve, it travels from the Pons to the external rectus across the floor of the cranial cavity. In its course it passes over the sharp upper border of the Petrous temporal and is fast at the cavernous sinuses and the Pons. According to one theory, if the intracranial pressure is increased there will be a tendency to press the brain back and down through the foramen magnum since this is the only large opening in the cranial cavity. If this is true, a tumor, cerebral hemorrhage, or any other condition causing an increase in intra-cranial pressure, will displace the brain in this manner thus increasing the pull on the abducen's nerve. With an increase in tension on the abducen's nerve conduction may easily be affected where this nerve passes over the sharp edge of the Petrous temporal and makes an almost right angle bend.

The trochlear, or fourth cranial nerve, innervates the superior oblique muscle and has the longest intracranial course and in part of its course closely approximates the course of the third nerve. The third and fourth nerves are most frequently affected by enlargements of the pituitary or hardening of the cerebral arteries since they are in close approximation to these structures. The sixth nerve is not affected as quickly by pituitary enlargement since it is protected at that point of its course by the internal carotid artery. The third nerve paralysis will result in the loss of function mentioned earlier. The third nerve is a very large nerve considering the muscles which it supplies. It is beyond the scope of this paper to discuss the nuclei of the third nerve and its many ramifications. It is sufficient to say that the superior branch supplies the superior rectus muscle and the levator muscle of the superior palpebrum while the inferior branch supplies the internal rectus, inferior rectus, and inferior oblique, also sending a short branch to the ciliary ganglion which will affect the pupil and the ciliary muscle.

As to the cause of paralytic tropias, probably the best source of statistics is Bielchowsky. He reports the following statistics: 15 per cent of his cases are the result of trauma; syphilis accounts for about 30 per

cent of his cases in most recent years and a greater percentage in earlier years. Paralysis of congenital origin constitutes 14 per cent of his cases. Epidemic encephalitis accounts for another 14 per cent. He gives no statistics on the anomalies of the blood vessels and other acute infectious diseases but mentions that these may be a cause of paralysis. His statistics show 15 per cent of the cases belong to the group of unknown etiology even after careful examination of the possible causes. It is interesting to note in passing that he mentions an increase in trochlear nerve paralysis in recent years due to (in his opinion) the use of Killian's operation for relief of frontal sinusitis. Earlier today you were shown that the trochlear (fourth nerve) has a course in close approximation to the frontal sinus and, therefore, could easily be injured by this radical operation.

On the prognosis of ocular paralyses Bielchowsky reports spontaneous recovery in 38 per cent of his cases with the highest percentage of recovery in trochlear nerve paralysis with abductions paralysis next in line and third nerve paralysis at the bottom of the list. If spontaneous recovery does not occur in six months to a year, operation may be considered to alleviate diplopia in the primary position of the eyes and also for cosmetic reasons.

In concluding this paper it might be fitting to mention some of the cosmetic reactions which are encountered with paralytic tropias. In general, the patient with a paralytic tropia will move his head in the direction in which he encounters the greatest separation of the diplopic images. For example, with paralysis of the external rectus of the right eye a patient will characteristically turn his head to the right and thus minimize or eliminate all diplopia. With paralysis of the superior oblique, the head position may be more complicated. Here there may be a tendency to tilt the head toward one shoulder. For example, in right trochlear paresis the head may be tilted toward the left shoulder, thus minimizing the effect of the paralysis of the superior oblique muscle. In some of the trochlear paralysis types you may also find a simple turning of the head in the lateral meridian away from the side of the paralysis since in this position the superior oblique muscle will have a minimum depressor effect. This is particularly true in cases where the paralysis is relatively mild. In some cases, the head may be tilted forward or backward to take advantage of the convergence impulse on downward gaze or the divergence impulse on upward gaze depending on which function will have an alleviating effect on the angle of the tropia. All of these head movements can quite logically be explained when a thorough understanding of the action of extra-ocular muscles in the various positions of the eye has been mastered.

158 WASHINGTON AVE.
SOUDERTON, PENNSYLVANIA

RECURRENT HORDEOLA ASSOCIATED WITH MONOCULAR SUPPRESSION—A CASE REPORT*

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HISTORY

Miss H. H., age 11, presented herself for an eye examination with the following complaints. For the past one and one-half years she has had styes constantly, the right eye suffering the most. She also complains of almost constant lacrimation and infrequent temporal and parieto-temporal headaches. Her present correction is five months old.

The patient has had the usual childhood diseases. There have been no serious illnesses or accidents. The dental health is claimed to be good. Recently, because of the hordeola she has undergone a series of checkups to determine the cause and the tests were negative. She received a series of injections to combat the possibility of a "blood infection."

The patient is a student, receives rather good grades, but is forced to miss school a great deal because of the styes. She was recently away for a few weeks during which time she did no close work and had no styes.

EXTERNAL EXAMINATION

O.D. Acute conjunctivitis.

O.S. Small hordeolum left upper lid. One recently opened on lower lid.
Acute conjunctivitis.

O.U. Convergence to 7 inches.
Rotations and versions normal.
Pupillary reaction to light and accommodation normal.

OPHTHALMOSCOPY

Media clear. Discs are normal in color, margins well defined. The physiological cups are normal in depth. The blood vessels are normal in ratio, there is no arterio-venous notching. There are no hemorrhages or exudates throughout fundi.

ANALYTICAL EXAMINATION

Unaided V.A. O.D. 20/20. O.S. 20/20.

Present correction:

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*Submitted as a portion of the entrance requirements of the American Academy of Optometry. Received from the Examining Board, July 19, 1947. For publication in the December, 1947, issue of the AMERICAN JOURNAL OF OPTOMETRY AND ARCHIVES OF AMERICAN ACADEMY OF OPTOMETRY.

†Optometrist. Fellow, American Academy of Optometry.

O.D. +0.75 D. cyl. x 90. V.A.=20/20.
O.S. +0.25 D. sph. ⊂ +0.75 D. cyl. x 90. V.A.=20/20.

Ophthalmometer reading:

O.D. 0.75 D. WR 90/180.
O.S. 1.00 W. DR 90/180.

Retinoscopy.

O.D. +0.50 D. sph. ⊂ —0.37 D. cyl. x 180.
O.S. +1.00 D. sph. ⊂ —0.75 D. cyl. x 180.

Subjective:

O.D. +0.50 D. sph. ⊂ —0.37 D. cyl. x 180. V.A.=20/20.
O.S. +1.00 D. sph. ⊂ —0.75 D. cyl. x 180. V.A.=20/20.

Phorias:

1 prism diopter of exophoria at 6 M.
Vertical=orthophoria.

Fusion faculty:

Periodic suppression of the left eye manifested both on the stereo-orthopter and when taking the phorias with the Maddox rod.

DIAGNOSIS AND TREATMENT

A change in the right lens was prescribed along with orthoptic training to break down the periodic suppression of the left eye. In this case the writer believes that the hordeola were not the result of the refractive error, but rather the result of the faulty fusion faculty, since the patient has been suffering with this problem for a number of years.

As to the etiology of the hordeolum, May, in his book, "Diseases of the Eye," states, "Styes occur at any age, but are most common in children and young adults. They often appear in crops or series. They are frequently associated with blepharitis, a lowered state of health, anemia, and *uncorrected errors of refraction.*"

MacElree of his book, "Manual of Ocular Pathology for Optometrists," states, "Hordeola may occur at any age, but they seem to be most common in childhood and youth. They have a very marked tendency to occur in series. They are definitely the result of infection with a pus-producing micro-organism, which in a majority of cases is believed to be the Staphylococcus Albus. A local skin disease (as a result of uncleanliness) may be the actual source of invading microbes. Refractive errors (among other causes mentioned) especially those of the compensated hyperopic variety, are often responsible for their production. This is due to the excessive hyperemia produced by such conditions. The

blood engorged tissues act as a perfect medium for the growth of the micro-organisms and the itching paves the way for their entrance."

It is interesting to note that neither of the authorities mentioned state that a defect in the fusion faculty might be a cause for hordeola.

Orthoptic training was instituted and continued for two weeks. The initial treatments were as follows: a constant, poorly illuminated target for the right eye. The left eye was given a highly illuminated target with a three second flash, to view. The target for the right eye was blue, that for the left, red. The flash treatment was given for four hours (four treatments), following which the patient possessed perfect simultaneous vision. Training was then continued on third and fourth degree fusion until 100 per cent stereopsis was attained.

Since the patient has been discharged, she has had no styes, lacrimation, or headaches.

COMMENT

From the above it may be concluded that the formation of hordeola in this case was a result of the defect in the fusion faculty. Although the correction the patient was wearing was somewhat incorrect, it is felt that at one time she must have been wearing the proper correction, but still suffered from styes, leaving the periodic suppression as the probable cause.

SUMMARY

The main complaint in this case was "the constant formation of styes." The patient had been examined innumerable times in the past one and one-half years, both ocular and general, to determine the cause of the hordeola, but to no avail. A change in correction followed by orthoptic treatments for a periodic suppression of the left eye has kept the patient symptom free for the past two months.

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WILMINGTON, DELAWARE

ELIMINATION OF CYCLOPHORIA AS A FACTOR IN ASTIGMATIC CORRECTION*

J. R. Shreve†
Indianapolis, Indiana

The test for the presence or absence of cyclophoria is one which is usually ignored in the routine refractive case. It is one of those special tests taken when some special problem presents itself. Perhaps the reason for ignoring it in routine work is the inconvenience of its application and the difficulty of interpretation on the part of both the patient and the examiner. The use of Maddox rods before both eyes requires that the test be taken under conditions which are not always available, that is, a highly darkened room having a single spot of bright light with little or no extraneous light. Otherwise, the streaks of light created are relatively dim, and are easily confused with extra, unwanted streaks. The test can be made on most stereoscopic or haploscopic instruments by using special rotary targets, but there may be some degree of uncertainty as to the result because of the possible effect of prisms or mirrors upon the test and the effect of nearpoint instrumentation upon the patient. Added to these difficulties is the question as to the ability of the patient to properly interpret conditions of parallelism or of perpendiculars, an ability which is by no means always reliable.

When cyclophoria has been tested and determined to be present, we are immediately confronted with the question as to which eye is at fault, or to what degree each may have turned. This can be determined by the use of special equipment. Maddox and others around the turn of the century devoted considerable thought to this problem. Still, even with the most reliable instrumentation and the most accurate finding, nothing is ordinarily done about the condition, since the phoria is corrected by fusion, while cyclotropia implies a paretic condition which is beyond the scope of our practice. To sum up: added to the difficulties of applying the cyclophoria test and the question as to the reliability of the finding, is the fact that the presence of cyclophoria has little significance in the disposition of our routine case.

Cyclophorias may, however, be causing trouble for us in our most routine cases, by interfering with the proper determination of the axis

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*Read before the Indiana Chapter, American Academy of Optometry, Indianapolis, Indiana, October 8, 1947. For publication in the December, 1947, issue of the AMERICAN JOURNAL OF OPTOMETRY AND ARCHIVES OF AMERICAN ACADEMY OF OPTOMETRY.

†Optometrist, Fellow, American Academy of Optometry.

or axes of astigmatism. All of our tests for astigmatism are conducted under conditions of monocular vision. It is, then, possible that a cyclophoria manifesting itself during the monocular astigmatism test may cause some error in the axis of the correcting cylinder in one or both of the eyes, an error which is especially serious if the power of the correcting cylinder is moderately high.

A question may arise here as to whether or not a cyclophoria *does* manifest itself in an eye which is fixating alone. It is possible that field details surrounding the astigmatic chart are sufficient to hold the eye in perpendicular alignment. But it seems equally possible that when monocular attention is concentrated upon a more or less circular astigmatic chart, a torsional movement may be made in spite of a resulting obliquity of the surrounding field. We have no way of assuring ourselves that the patient could or would reliably interpret and report such obliquity, nor do we have any clinical method of measuring or correcting such obliquity. This question might be answered by experimental methods not available in routine practice, but we will find that it is much easier to eliminate the possibility than to worry about the question.

In order to eliminate the possibility of the cyclophoria's manifesting itself in sufficient degree to disturb the axis determination, it is necessary to allow the eye *not* under test sufficient peripheral vision to hold binocular fusion, and at the same time eliminate that eye's view of the astigmatic test chart. This can be accomplished by making minor changes in equipment and slight adjustment in refractive routine without adding materially to the time or effort of that routine.

The method we use involves color filters. Using a Clason projector, a red filter (Eastman Wratten gelatin filter number F29) is incorporated into the slide of astigmatism test figures so as to provide a red background for those projected figures. This also provides such advantages as may be present in the use of monochromatic red light. The Robinson-Cohen astigmatic cross also provides a red field which has been found to work perfectly for our purpose. For charts other than those which are projected it may be possible to obtain the same effect by placing a large filter, such as Kodaloid acetate number 0075, in front of the astigmatic chart.

Now, a red-free filter (greenish-blue) is placed in the aperture before the eye *not* under test. We refract with some degree of light in the room, and this is ordinarily sufficient to allow the eye behind the greenish-blue filter enough vision of the distant field surrounding the screen to provide binocular fusion. At the same time, this greenish-blue

filter absorbs the monochromatic red light so that the eye not under test is prevented from seeing the astigmatic chart on the screen. This greenish-blue filter was obtained as special accessory equipment with the Green refractor. Such a filter is available from any of the large ophthalmic manufacturers, or it may be adapted in the office by using Eastman Wratten gelatin filter number 44.

There is little doubt that similar results would be attained through the use of properly placed polarizing elements. In fact, it is probable that the use of such elements would be even more effective, since the amount of light reaching the two eyes from the field surrounding the test chart would be more nearly equal. We have not inquired as to whether the polarization of projected astigmatic charts might adversely affect the astigmatism test.

SUMMARY

A method is described of testing the astigmatism of one eye with a monochromatic red chart, while the other eye is denied a view of this chart by a red-free (greenish-blue) filter but is allowed sufficient vision of the surrounding field to provide binocular fusion. In this way it is believed that we eliminate the possibility of a latent cyclophoria manifesting itself so as to cause error in the axis of the astigmatic prescription.

709 EAST 38TH STREET
INDIANAPOLIS, INDIANA

CORRESPONDENCE

TILLYER FUL-VUE TRIFOCAL INTERMEDIATE LENS VALUE

To the Editor:

In Volume 24, No. 8, of the AMERICAN JOURNAL OF OPTOMETRY AND ARCHIVES OF AMERICAN ACADEMY OF OPTOMETRY there appears an article on "Trifocals—Their Theory and Application," (pp 359-367). This is an excellent article, but we wish to correct the statement that the Tillyer Ful-Vue trifocal as manufactured by the American Optical Company has a 66 per cent intermediate. The AO Tillyer Ful-Vue trifocal intermediate has a power of 50 per cent of the (reading) addition rather than 66 per cent intermediate as mentioned under table one. The intermediate of a + 2.50 D. addition is + 1.25 Diopters, instead of + 1.66 Diopters as listed in Table II.

E. WILLIAM TILLYER

NOTES ON AMETROPIA—A FURTHER ANALYSIS OF STENSTRÖM'S DATA*

Monroe J. Hirsch, Ph.D.,† and Frank W. Weymouth, Ph.D.‡
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Stenström[1] in 1946 made careful measurements of three of the more important variables of the human eye and also of the state of refraction. This work recently has been discussed by Stoddard[2] in this journal, and is as he points out "a tremendous contribution to our knowledge of the refractive components of the eye . . . and . . . the greatest step forward in this field since the days of von Helmholtz and Gullstrand." It is the opinion of the present authors that this is true and in addition that much valuable information can be obtained by further analysis of Stenström's data; it is the purpose of this paper to present certain computations not appearing in the original paper nor in the review mentioned above. The primary data dealt with in the calculations that follow are those of Stenström.

Essentially, Stenström made four determinations on each of 1,000 right eyes. These measurements were of the axial length (b) following the X-ray technic of Rushton;[3] the radius of curvature of the cornea (c) by use of a Javal-Schiötz keratometer; the anterior chamber depth (d) according to the method of Lindstedt;[4] and the total refractive state (A) or static refraction by the methods of Donders.[5] It should be stressed that as far as these data go, the four variables were measured directly and independently and no assumptions were necessary as in former work along these lines. Stenström also presents and treats such

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1. Stenström, Sölve: Untersuchungen über die Variation und Kovariation der optischen Elemente des menschlichen Auges Acta Ophthalmologica, Supplementum xxvi, Copenhagen, 1946.
2. Stoddard, Kenneth B.: Myopia—the Present Status. Am. J. Optom. & Arch. Am. Acad. Optom. *24*. 313-320, 1947
3. Rushton: Trans. Ophth. Soc. U. K. *58*: 136, 1938.
4. Lindstedt: Am mätning av fr. ögonkammerens djup.. 1913.
5. Donders, F. C.: Die Anomalien der Refraktion und Akkomodation des Auges Wien, 1866.

variables as power of the lens and total refractive power of the globe. To determine these, however, he used the data of the four elements which he measured directly. Hence, correlations involving either of these calculated factors would by virtue of having been determined from the other measured quantities be in part spurious. For example, the correlation between lens power and state of refraction would be influenced by the presence of a common element since the measured state of refraction was used in the calculation of the lens power. In the analysis here presented, however, this difficulty has been avoided by limiting the discussion to the four elements which were, as has been stated, determined independently and by direct measurement.

The six coefficients of correlation which Stenström gives, hereafter referred to as primary and which are taken as the basic data for this study are presented in Table 1.

TABLE 1—STENSTRÖM'S PRIMARY COEFFICIENTS OF CORRELATION

Refractive State—Axial length	A b	—0.76
Refractive State—Corneal radius	A c	+0.18[6]
Refractive State—Anterior chamber depth	A d	—0.34
Axial Length—Corneal radius	b c	+0.31
Axial Length—Anterior chamber depth	b d	+0.44
Corneal Radius—Anterior chamber depth	c d	—0.10

The coefficients of correlation are measures of the covariation of the two elements concerned. The other two variables are present, however. It is often desirable to get rid of the effect of the other variables. Thus, for example, we have seen that the correlation coefficient between axial length and state of refraction was —0.76. This, however, includes cases with a wide range of corneal radii. Suppose now, that we wished to know what the correlation would be for just a single value for radius of curvature of the cornea. Obviously we might obtain this information

6. Unfortunately, Stenström used refractive power of the cornea in some of the correlations and radius of curvature of the cornea in others. To utilize the data it was thus necessary to arrive at the correlation between A and c which is not given in the original paper. However, Stenström does give the correlation between refractive state and corneal refractive power as —0.18. Since the corneal power is related to the radius as follows:

$$\text{Corneal Power} = \frac{n - 1}{c} = k \cdot \frac{1}{c}$$

the following is true: a) since the same constant k is used for all subjects, the numerical value of the correlation coefficient will not be altered since multiplying a variable by a constant does not change relative value of this variable; b) the use of the reciprocal will change the sign of the coefficient from — to +; c) the use of the reciprocal will change the value of the coefficient of correlation but slightly. The magnitude of this last is not known, but it is probably small. Thus the correlation A c which Stenström did not present and which was necessary in this paper has been assumed from the correlation between corneal power and A. This coefficient being —0.18, the assumption has been made that the correlation A c would not be far from +0.18.

by limiting the cases involved in our correlation to those whose corneal radius was of a given magnitude. This would be an experimental control, and clearly would necessitate the measuring of a great number of individuals to discover the smaller number who would be used in the correlation. The same effect may, however, be obtained statistically, by holding certain of the elements constant, the technic being called partial correlation.[7] The correlation coefficient of —0.76, given above, is that between state of refraction and axial length for a wide range of corneal radii. Partialling out the corneal radius element raises this coefficient to —0.87. Thus we may say that if we were dealing with a population in whom the corneal radius was constant instead of variable, the axial length would play a far greater part in determining the state of refraction. A useful interpretation of any correlation coefficient is that the variance in one variable due to variability in the other is as the square of the coefficient. Thus, in the above example, we may say that the variance in state of refraction due to the variance in axial length is 52 per cent (.76 squared), but that were it not for variability in corneal radius, this would be 76 per cent (.87 squared).

Just as it is possible statistically to partial out a single element, so it is possible to partial out two or more. Thus we might determine what the correlation between state of refraction and axial length would be if anterior chamber depth as well as corneal radius were held constant. Such partial correlation coefficients have been calculated by the present author and are presented in Table 2. In each case the two measured elements not appearing in the correlation have been partialled out. The effect of holding constant the elements not being considered may be seen by comparing these coefficients with those primary correlation coefficients already presented in Table 1.

TABLE 2—PARTIAL CORRELATIONS CALCULATED FROM STENSTRÖM'S PRIMARY CORRELATION COEFFICIENTS

Refractive State—Axial length	A b	—0.87
Refractive State—Corneal radius	A c	+0.70
Refractive State—Anterior chamber depth	A d	+0.25
Axial Length—Corneal radius	b c	+0.75
Axial Length—Anterior chamber depth	b d	+0.46
Corneal Radius—Anterior chamber depth	c d	—0.37

As has already been mentioned, Stenström in the original paper did present a few partial correlations, statistically holding constant a single element. However, for some reason not known to the present authors, he did not systematically calculate the complete set of partial correlations presented in Table 2.

7. Stenström was aware of the technic and calculated a few partial correlations. However, he neglected to calculate and present the set of partials which form the basis for the present paper.

DISCUSSION

The correlation between refractive state and axial length gave the highest primary coefficient, —0.76. When the other two variables, corneal radius and anterior chamber depth, were held constant this coefficient increased to —0.87. Stoddard, in his review of this data, pointed out that, "it would appear that most myopia is axial in nature." The partial correlation demonstrates that not only is this true, but to an even greater extent than was demonstrated by the primary correlation. There can be no question but that axial length is by far the most important of the variables, and that variance in state of refraction is attributable to this element more than to any other.

The primary correlation between refractive state and corneal curvature was quite low, being +0.18. However, when axial length (which we have just shown to be the strongest determinant of refractive state) and also anterior chamber depth are partialled out, this coefficient increases to +0.70. This change between primary and partial, the second largest obtained, demonstrates that corneal curvature is an important determinant of refractive state, but because it does not play as great a part as axial length, the fact was obscured in the primary correlation. The primary and partial correlation between corneal curvature and refractive state clearly indicate that while corneal radius is not as important as axial length in predicting state of refraction, nevertheless, other things being equal, corneal curvature could account for a considerable part of the variability in state of refraction.

The primary correlation between refractive state and anterior chamber depth was —0.34. The interpretation of this negative correlation would be that a small chamber depth is found associated with hyperopia. This, however, seems strange since the cornea and lens each contribute to the total refractive power of the eye, and increasing the distance between two lens components of an optical system like that of the eye has the effect of decreasing the total power. Thus, increasing the chamber depth should make for hyperopia, yet we find that the primary correlation is interpreted as meaning that hyperopia and a small chamber are associated. Now, when the partial correlation is considered, it is striking, indeed, that this and only this coefficient showed a change in sign. The partial correlation of +0.25 may be interpreted as meaning that other things (particularly axial length) being equal, a large chamber depth is associated with hyperopia. This, as was shown above, is what we would expect on the basis of the optics involved. Before attempting further interpretation, it would be well to consider the corre-

lation between axial length and anterior chamber depth. This, in the primary data was +0.44 and was increased slightly to +0.46 in the partial. Thus in either case, the easily understood fact is that the longer the total length of the globe, the greater the distance between cornea and lens. Since the depth of the chamber is part of the total length, we would be, indeed, surprised if this were not the case.

Returning now to the correlation between chamber depth and state of refraction, we may reach the following interesting and important conclusions. Increased axial length is associated with, and undoubtedly is a major cause of myopia. Furthermore, as might be expected since growth tends to be harmonic, the longer the eyeball, the greater the depth of the anterior chamber. As a result of these two facts, it would be expected that associated with myopia would be a larger chamber. This is what the negative sign of the primary correlation would indicate. However, from the optics of the situation we know that the effect of a large chamber, increasing the distance between the two positive powered refractive surfaces would be to decrease total refractive power and cause hyperopia. The partial correlation demonstrates that this is true, and when we rid ourselves of the effect of other variables, this is the case. Here, then, is an emmetropization mechanism *par excellence;* increased axial length causes myopia . . . but it also is associated with an increased anterior chamber which in turn causes a decrease in myopia. On the basis of this alone, we may say that harmonic growth causing the anterior chamber to become deeper as axial length increases has probably accounted for more "reduction of myopia" than all of the numerous procedures that have been suggested in the past as cures for this type of ametropia.

Another emmetropizing mechanism, and one of perhaps greater importance is found when we consider the correlation between axial length and corneal curvature. Stenström mentions the theory of Bourgeois and Tscherning[8] of "harmonic growth." They noted that the corneal radius was usually one-third the magnitude of the axial length. Confirmation of such a theory would require a positive correlation coefficient between these two variables. The primary correlation coefficient has been given in Table 1 as +0.31. However, holding state of refraction and chamber depth constant increases this to +0.75.

Logically, this high correlation between corneal curvature and axial length is to be expected. An eyeball large in one meridian might well be expected to be large in the others. It therefore follows that an eyeball

8. Bourgeois and Tscherning: Ann. d'ocul. 96: 203, 1886.

that was long would also be one with a large radius of curvature of the cornea. The former circumstance would tend to make the eye myopic, the latter, hyperopic. Here again, then, is evidence not of combination of the elements of the eye in a random fashion, but rather in harmony.

The fact that axial length was the most important determinant of refractive state was discussed above. There are other approaches to the problem of relative importance of the various optical elements here considered. One such approach is that of multiple regression. If one were to predict the state of refraction from knowledge of the axial length, corneal radius, and depth of the anterior chamber, the formula would be one of multiple regression. In this, each factor would be multiplied by an appropriate coefficient and when these were summed the total would give the state of refraction. The coefficient by which each factor was multiplied would represent its contribution to the total state of refraction. These "standard partial regression coefficients" obtained in a manner similar to the partial correlation coefficients above, are given in the following table. These may also be combined into a single multiple correlation coefficient, R, which represents the total amount of any variable, here the state of refraction, that is accounted for by the other factors being considered. For this data, the value of R is +0.885, and as already mentioned, the square of this, .783 indicates the percentage of "explained" portion while 21.7 per cent of the variance depends upon factors other than the three here used.

TABLE 3—PER CENT OF VARIANCE IN REFRACTIVE STATE DUE TO VARIANCE IN THE THREE MEASURED FACTORS

Factor	Coefficient	% Explained
Axial Length	—0.975	47.42
Corneal Radius	+0.496	24.13
Anterior Chamber Depth	+0.138	6.74
Unexplained Factors	—	21.71
		100.00

Thus, approximately 47 per cent of the variance in refractive state is due to axial length, 24 per cent to corneal radius, and about 7 per cent to chamber depth. The remaining 22 per cent must include variability in crystalline lens curvature, indices of refraction of each of the media, and errors of measurement. Stenström's correlation between lens power and refractive state was zero. Since the calculation of lens power did, however, depend upon the refractive state, this correlation may be partly in error. The multiple regression, however, also shows that if the lens makes any contribution to the total refraction it is small, since the 22 per cent of the variance which must be divided between lens power, indices of refraction, and errors of measurement. Even if all of the re-

maining total amount which is unexplained were due to the lens, its contribution would be still less than one-half that of the axial length, and no greater than that of the cornea.

There is still another way to determine the effect of each of the three variables on the refractive state. Stenström determined the mean and standard deviation for each of the variables. By geometric optics the possible effect of extreme deviations in the variables can be calculated. Thus, three standard deviations could be added to the mean of the depth of the anterior chamber, giving the maximum depth to be expected, and the effect of such a change on the state of refraction calculated. This process could be repeated with the mean minus three standard deviations, giving the minimum depth of chamber. The difference between these two values would give the extreme effect on total refraction of the greatest change in chamber depth that might be expected. Similar calculations might be made for the effect of axial length and for corneal radius.

Such calculations indicate that extreme variations in the chamber depth would make a change in the refractive state of 1.1 D.; in the corneal radius, a change of 7.7 D.; in the axial length a change of 17.1 D. Expressing the first two as percentages of the latter, we find on the basis of the original frequency distributions, that the possible range in the depth of the anterior chamber would account for only 6 per cent as much as the axial length, while the corneal radius could account for 45 per cent as much. If these values are compared with the results previously described, it is found that the method of multiple regression indicates that corneal radius accounts for 51 per cent and chamber depth 14 per cent as much effect as the axial length. Similarly, if the squares of the partial correlation coefficients are compared, the values are 65 and 8 per cent.

The close agreement of these three approaches is striking. On the basis of the three sets of information it may be said that about half of the variance in the refractive state is due to variation in axial length, and that the importance of the cornea is about half of this or one-fourth of the anterior chamber one-tenth of the axial length, or about one twentieth of the total. The remainder, about 20 per cent, is due to all other sources of variation, including refractive indices, refractive effects of the crystalline lens and errors of measurement.

SUMMARY

Additional calculations have been made, based upon the excellent data of Stenström, use being made only of the four independently de-

termined variables, refractive state, corneal radius, axial length, and anterior chamber depth. These calculations include partial correlation coefficients, multiple regression coefficients, and the effect on the refractive state of the extreme range of each of the other variables as derived from the original frequency distributions. On the basis of these calculations the following modifications and additions to the conclusions of Stenström are indicated.

(a) The cornea plays a much more important part in determining the refractive state, i.e., ametropia, than suggested by the primary correlations presented by Stenström. The still greater importance of the axial length tends statistically to obscure the effect of the corneal radius.

(b) The three elements are correlated, i.e., do not combine in random fashion. In general, the longer eyeball is associated with a less refractive cornea (greater radius) and a deeper anterior chamber. The effect of the corneal and chamber growth is such as to oppose the effect of increased axial length, thus tending toward the production of emmetropia.

(c) The effect of the range of corneal radius and chamber depth is, however, far less than that of axial length. Thus, while the relations are such as to tend to emmetropize the eye, the axial length remains the most important factor.

(d) About half of the variance in the refractive state is due to axial length, a fourth to the corneal curvature, and a twentieth to the depth of the anterior chamber. The remaining fifth of the variance in the refractive state must be divided between variations in the crystalline lens, refractive indices, and errors of measurement.

DIPLOPIA RESULTING FROM PARESIS OF THIRD NERVE CAUSED BY FOCAL INFECTION—A CASE REPORT*

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Patient: Mrs. S. K., age 24. This patient called June 26, 1945.

Her past history showed a good general health condition with no hereditary diseases. Upon inquiring into her present health,. I found that her teeth were in fair condition with very few cavities; and that she was told repeatedly over a period of years that her tonsils were bad and should be removed.

Her complaint was, "About one and half months ago, I suddenly saw things double and noticed my eyes were crossed. Approximately at this same time I had an attack of tonsillitis. I have no headaches or pain, although bright light hurts my eyes. This all makes me very nervous and I need relief badly."

Her visual acuity was 20/15 O.U. The cover test showed an exotropia of approximately 25 degrees.

An external examination revealed no pathology. The versions of the eyes showed a possible paresis of the internal recti as the eyes could not move in. The right eye moved only as far as the midline, while the left eye moved slightly inside the midline. The pupils were larger than normal and their reaction to light was slow and sluggish. The writer noted that upon looking at her tonsils they were red and swollen, hardly allowing any sort of passage down the throat.

An ophthalmoscopic examination showed a good normal fundus: disc of good color and definition; the vessels were in their proper relation with one another; the media were clear.

The manifest and subjective findings showed a negligible refractive error: O.D.+0.75 D. sph. ⌒ +0.25 D. cyl. ax 90. O.S.+0.50 D. sph. As far as could be determined with rotary prisms, she had a 26 diopter exotropia and a 13 diopter right hypertropia. Her amplitude of accommodation was low for her age: it was 4 diopters in O.D., and 5.5 diopters in O.S.

A prismatic correction, which was not ordered, allowed her to see singly. It was O.D. 15△ base-in ⌒ 4△ base-down. O.S. 4△ base-in

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⌒ 8△ base-up. Her fields were taken and found to be quite normal, with a possible exception in the right eye, the constriction being in the temporal field.

Then tests of neumonics were taken and to the best of my knowledge I ascertained that the patient had a paresis of the right internal rectus and a paresis of the right superior oblique. In the left eye the patient had a paresis of the left internal rectus. Thus, an involvement of the III and IV Cranial nerves, this probably due to the toxic affect of the focal infection from the tonsils.

The writer recommended that the patient wear a patch over one eye so as not to be bothered by the diplopia; and to see her physician immediately about removing her tonsils. She was told to return a week after the removal of the tonsils.

The patient returned July 13, 1945; a week after the removal of the tonsils. Her condition was unchanged, diplopia, etc. Patient was told to return in two weeks.

The patient returned July 27, 1945, three weeks after the removal of tonsils, and this time reported no diplopia or visual fatigue of any kind.

Her phorias at far and near were higher than normally found, and there were only 4 diopters of right hyperphoria. The amplitude of accommodation was now 9 diopters O.U. There still was present a very slight paresis of the left internal rectus noted upon having patient look to the extreme right. No correction was given, and the patient was told to report back in two months for a final check-up.

Several weeks later the patient called and reported that she once again had diplopia and the eyes were crossed again. I then asked the patient to see an ophthalmologist. This was done.

His report follows: "Mrs. S. K.'s eyes were thoroughly examined and a paresis of her left third nerve was found. She has diplopia in practically all fields. She undoubtedly has a neuritis of the nerve as an effect of her recent tonsil infection. It would be best, as a temporary expedient, to block off one eye. In treatment, I am suggesting twice weekly injections of 10,000 units of thiamine, and three capsules daily of becotin."

The patient returned recently and her diplopia is practically nonexistent except on extreme rotation of eyes.

COMMENT

This type of case is uncommon but not rare. It shows that there is a definite relationship between ocular complaints, visual or otherwise, and systemic or focal infection. It calls for an awareness, on the optometrist's part, of a general recognition of diseases and deformities other than that found in the eye itself.

THE TOLERANCE FACTOR AND SATTLER'S VEIL AS INFLUENCED BY A NEW DEVELOPMENT OF CONTACT LENS MAKING*

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From the time contact lenses came into fairly general use it has been universally recognized that their main limiting factors are: (a) Their variable wearing time, i.e., their tolerance. (b) Sattler's Veil, sometimes known as Fick's phenomena.

The modern types of contact lenses with their changes in form and construction have little influenced these twin limitations. It has been established generally that with all these variations (confined mainly to changes of construction in the haptic) these restrictions are ultimately present, although in a few instances patients have given indications of tolerances well above the average. These shortcomings prompted the writer, from 1943 onwards, to carry out experimental research into their causation.

Other writers have indicated alterations in the composition of the fluid lens produce changes and delay in the onset of Sattler's Veil and its inter-related tolerance factor. Patients usually preferred a particular strength and composition of the fluid and this had to be determined by trial, although quite a number of cases achieved equality of tolerance with a fairly wide range of fluids.

Many strengths and compositions of buffer solutions have been tried by the writer, with varying results, some causing the appearance of Sattler's Veil long before the contact lenses began to feel uncomfortable. Other recordings indicate the converse and in further cases the onset of the Veil and cessation of tolerance was simultaneous. With all patients, however, the onset of Sattler's Veil was noticed after a certain period, and the limitation of tolerance experienced. Sattler's Veil may be defined as a combination of mistiness of vision plus the appearance of rainbow-colored halos seen round naked lights. It has been observed that the chemical composition in the types of contact lenses in present day use is of prime importance and initially the writer held the opinion

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that the solution of the problem lay in the changes of the strength and composition of the fluid.

Early in the experimentation the theory was formulated that the very tiny crystals sometimes found in the residual fluid of the contact lens solution were the cause of the veil. By their very nature in pattern they could either act as a diffraction grating or as a series of very small prisms causing the halos and mistiness. When a contact lens was worn for a considerable period, the stratified epithelium of the cornea was somewhat coagulated and it was thought that a residue of these crystals lodged in the coagulations for some time even after the contact lens had been removed. Microscopic examination and chemical analysis of these tiny crystals showed that they were rich in chlorides, but that they did not readily dissolve in water. The indication was therefore that these must have been crystals of the tear fluid which had penetrated the buffer solution; crystals of the tear fluid are rich in insoluble chlorides (ref. Lipschutz and Ahlstrom) but by their very nature the crystals of the sodium chloride buffer solution used are readily solvent. At this stage the writer thought that all that had to be done was to use a fluid which could resist the penetration of the tiny crystals of the lacrimal fluid. Various emollients were tried but with disappointing results, in effect the normal buffer solutions gave much better results.

In some patients, after contact lenses had been worn for a minimum of two to three hours, a slight coagulation of the stratified epithelium of the cornea was observed under the slit lamp, using the highest power O.G. available. It was thought that this might possibly be the cause of the veil. Artificial coagulation of the corneal epithelium in a normal patient who had not worn contact lenses previously failed to produce any veiling.

It would be going over well trodden ground to repeat my tabulated relevant details of the various experimental solutions tried on patients, as these details do not produce any essential deviation from published findings. Nevertheless, it may be of general interest to record that of all the solution used the most satisfactory method of delaying the onset of Sattler's Veil and of increasing tolerance was by the following use of sodium bicarbonate solution. One per cent sodium bicarbonate solution was used as the buffer solution and the lenses worn until symptoms of the veil became imminent; the lenses were at once removed and immediately re-inserted with 2 per cent solution. Once again the lenses were worn until the veil became imminent when sub-

stitution for 3 per cent solution was found to prove efficacious, this practice is capable of extension into higher concentrations, thus an appreciably greater tolerance was achieved by this "stepping up process," after other solutions had in comparison given less satisfaction.

In plotting variations of the tolerance factor against various types of scleral fittings a most irregular curve resulted and no constant achieved. It was noted, in some cases, that poor scleral fitting gave a reasonably good tolerance. As a general rule, however, a well fitting lens gave the greatest all round comfort. A closer relationship was established in plotting variations in the limbal fit against the tolerance factor and although no constant was derived the curve plotted proved more steady. The indications from these experiments showed that a 1 mm. limbal clearance and certainly not more than 2½ mm. clearance produced the most satisfactory results. In all these instances full apical clearance was given. Generally the results showed that an absolute scleral fit gave a greater tolerance than a lens modified from this extreme, and in particular some improvement was observed where loose areas or shallow channels seemed to extend to the periphery of the lens without breaking the vacuum seal. A lens inserted purposely with a bubble definitely produced a longer tolerance and delayed the onset of the veil.

Having regard to results obtained to date, the writer felt that if it were possible to do away with the artificial solution used and replace it by a normal flow of lacrimal fluid and in addition permit air to enter the hitherto enclosed chamber between the anterior surface of the cornea and the posterior surface of the contact lens, many of the difficulties pertaining to these lenses would be improved if not entirely solved. An additional advantage would accrue from permitting a free interchange of oxygen from the air and dispersal of carbon dioxide. The author has therefore developed a new type of contact lens in which certain defects attaching to known contact lenses are obviated or mitigated. At present, there is provided a contact lens with a plurality of holes, situated in the scleral or corneal portion or at the circum-corneal junction. These holes are provided to permit lacrimal fluid and/or air to flow through the lens while in position on the eye. The holes may be arranged in spaced, single or staggered relation one to another (they may be staggered radially) in circular, or arcuate rows. The apertures may be in the form of slots, or in the form of circular or triangular or substantially rectangular cross-section. Preferably the angle of the upper holes (under the upper eyelid) are substantially vertical and the lower holes (those which lie principally under the lower lid) are substantially horizontal. The vents may have

their ends rounded or slanted so as to conform with the direction of flow of the lacrimal fluid.

With regard to the fitting of these lenses the improved contact lens is so mounted as to provide for a *permanent* presence of flowing lacrimal fluid over the entire pupillary region, thus filling up and neutralizing any corneal irregularities. The contact lens must be fitted sufficiently close to the cornea to provide for such filling and neutralization. A primary feature of the construction is that it permits a flow of lacrimal fluid and/or air through the lens while in position. The lower apertures constitute vents to provide a drainage means for the lacrimal fluid in such a manner that the fluid flows substantially along the normal course it would follow if the contact lens were not fitted. In order to ensure this, the number, slope, position, angle, shape, direction and size of the upper and lower holes may be varied within relatively wide limits for providing a satisfactory means of flow and egress of the fluid to and from and thus through the lens. Slots and ducts may be conveniently substituted for the mentioned apertures and holes in the adaptation of this principle.

Fitting techniques are the same as practiced at present with the exception that minimum apical clearance must be achieved so that the posterior contact lens surface approximates in curvature to the curvature of the cornea for at least over an area of 2 mm. beyond the maximum regular pupillary diameter. In practice this ideal has proved most difficult to attain and due care must be taken to observe and allow for the "settling down" effects when the lens has been in position for some time.

To summarize, some of the observed benefits include, a constant change of fluid at the rear of the lens, and provision for the access of air, i.e., atmospheric oxygen to enter and carbon dioxide liberated. No preselected buffer solution is required, and Sattler's Veil together with the tolerance factor will be either eliminated or very favourably influenced. Asphyxia is obviated and the suction effect greatly mitigated.

Some experimental patients already wearing contact lenses were re-fitted with these modified lenses and a marked increase in tolerance was at once noted. With all the cases experimented the colored halos did not appear at all—in some instances a slight mistiness occurred after several hours wear. It was generally reported that the contact lenses could have been tolerated indefinitely in contradistinction to their former limited tolerance. It is felt that in the cases where mistiness occurred, additional research and subsequent modification of the vents would have improved these results further. It was found that the size and number

of the holes as well as their distribution showed a marked influence in postponing the onset of the mistiness. One case recorded had one vent in the upper and one in the lower portions of the lens, showed a marked improvement when three holes were incorporated in their respective hemispheres.

A typical example is that of a young optical student who with a well fitting orthodox lens obtained a maximum tolerance of 5 hours continuous wear with comfort, although by the end of this period an appreciable Sattler's Veil was present; upon being refitted with the modified lens he obtained practically indefinite tolerance. On one occasion he wore his lenses for nearly 16 hours. On two occasions the patient was able to wear his lenses without mistiness and on two other occasions some mistiness was present, but on all occasions the colored halos were absent.

A case seen by a colleague in the United States is now able to wear his lenses with comfort all day whereas his previous tolerance was strictly limited.

Whilst the writer feels that at this early stage definite claims cannot be made it should, however, be borne in mind that although a measure of success on the limited number of experimental patients has been achieved the full value of this development cannot be accurately assessed until it has been tried out in practice on a large number of patients.

The research work which has been carried out throughout these years, and which has prompted me to patent this development, has been made possible by the constant help and encouragement which I have received from my friend and colleague Mr. P. J. Cole. In addition my appreciation must be expressed to Messrs. E. F. Fincham, S. W. Dunn and in the United States to Drs. Silverstein and Greenspoon who have undertaken to carry out independent research on this development. My grateful thanks must also be expressed to Messrs. C. W. Dixey who were instrumental in giving me facilities for my initial experiments and in making the first modified lens. It would be unfair to conclude such a paper as this without recording acknowledgments to those workers who have stressed the desirability of permitting free entry of air and lacrimal fluid into the retro contact lens chamber. To their foresight full credit must be given.

37 MANCHESTER STREET
LONDON, W. I. ENGLAND

VISUAL TRAINING AS AN ADJUNCT TO REFRACTION
A CASE REPORT*

Sidney S. Haniford†
Buffalo, New York

CASE HISTORY

The patient, Miss J.S., age 11, a sixth grade pupil, came in for a visual examination upon advice of school nurse. Patient complained of headaches after movies, and of having to hold reading material very close to her eyes. Patient said that after a short period of reading, print became blurred. When given a book to read, she brought same to six inches from her eyes.

HEALTH HISTORY

Patient was in good health. She had had a medical examination a short time prior to her visit to writer's office. Her tonsils had been removed, and her only early childhood illnesses were measles and chicken pox from which she had recovered fully with no after effects.

PRELIMINARY FINDINGS

Patient seemed slightly nervous, especially with parent in the room. Father was then asked to leave refraction room but still the best unaided visual acuity was O.D. 20/100. O.S. 20/70.

External examination: Sluggish pupillary reflexes; lid margins clear; slight conjunctival injection; cornea clear.

Ophthalmoscopic examination: Media clear; discs well defined; vessel distribution normal; fundus slightly pale.

Static retinoscopy: Small amount of plus in both eyes recognized at once as being entirely out of agreement with the unaided visual acuity. Patient was given pair of +0.75 spheres to be worn at all times unless the blur was too annoying, in which event they were to be worn only for all close work. With the +0.75 spheres, patient's distance visual acuity was 20/70 O.U.

Patient was told to report back in two days. At this time her vision with the +0.75 spheres was 20/50. She was started immediately on visual training, with emphasis on forcing plus acceptance. She was also given a blinder to be worn three hours over the right eye, and three hours

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†Optometrist. Fellow, American Academy of Optometry.

over the left, alternately. After five training periods, a complete visual analysis was made with the following analytical findings.

ANALYTICAL EXAMINATION

Ophthalmometer

O.D. 42.50 at 180 43.75 at 90
O.S. 42.25 at 180 43.50 at 90

Static Retinoscope

O.D. +0.50 D.sph. ⊂ —0.50 D.cyl.axis 180
O.S. +0.25 D.sph

Subjective

O.D. +0.75 D.sph. ⊂ —0.50 D.cyl.axis 180
O.S. +0.62 D.sph. ⊂ —0.37 D.cyl.axis 180

Induced phoria: 4 exophoria

True Add.: 10D

Convergence: 19/0

Abduction: 10/8

Vertical Phoria: 0

Induced Nearpoint Phoria: 10 exophoria

Unfused cross cylinder

O.D.+1.25 D.sph. ⊂ —0.50 D.cyl.axis 180. Gross
O.S. +1.00 D.sph. ⊂ —0.37 D.cyl.axis 180. Gross
Induced phoria: 12 exophoria

Fused cross cylinder

O.D. +2.00 D.sph. ⊂ —0.50 D.cyl.axis 180. Gross
O.S. +1.75 D.sph. ⊂ —0.37 D.cyl.axis 180. Gross
Induced phoria: 14 exophoria

Tolerance adductive stimulation: 18 D

Positive fusional reserve: 25/18

Tolerance adductive inhibition: 15 D

Negative fusional reserve: 28/24

Amplitude of accommodation: O.U. Plus 3.75

Tolerance of accommodative stimulation: Minus 1.75

Tolerance of accommodative inhibition: Plus 2.25

The following prescription:

O.D. +1.00 D.sph. ⊂ —0.50 D.cyl.axis. 180
O.S. +0.75 D.sph. ⊂ —0.37 D.cyl.axis 180

was prescribed and visual training continued with emphasis on building accommodative rock and amplitude. After eight training periods, *amplitude was 7½* D, and vision with above prescription was *20/20 O.U.* Patient was now reading at 11 inches with no tiring of eyes or blurring of print. Progress report three months later showed complete comfort with all skills, taken on telebinocular 100 per cent and *amplitude 7.75 D.* Two years later, unaided visual acuity was 20/20 O.D., O.S., and O.U. Prescription was changed to:

O.D. +0.50 D.sph. ⌒ —0.50 D.cyl.axis 180
O.S. +0.50 D.sph. ⌒ —0.50 D.cyl.axis 180

CONCLUSION

This case illustrates the care required in taking visual acuity findings in school age children. We believe that if Miss J. S. had not been given corrective optometry in time, she would have developed into a typical student myope. The case also shows that in this type of problem, a complete examination should very often be delayed for three or four visits.

SUMMARY

By delaying a complete refractive examination until patient overcame apparent nervous tension, and by administering corrective optometry in the form of orthoptics, Miss J.S., age 11, was given normal visual acuity for distance with comfort and efficiency at reading. Timely preventive optometry thus saved the individual from possible school myopia.

289 WEST FERRY STREET
BUFFALO, NEW YORK

ERRATUM

In the paper, "An Investigation of the Use of Stereoscopic Targets in Orthoptics" by Dr. Meredith W. Morgan, Jr. AMERICAN JOURNAL OF OPTOMETRY AND ARCHIVES OF AMERICIAN ACADEMY OF OPTOMETRY, Vol. 24, No. 9, pp. 411-432, the final reference, (No. **24), lists the wrong publication.** This reference should read, Wald, G. & Burian, H., The Dissociation of Form Vision and Light Perception in Strabismic Amblyopia. American Journal of Ophthalmology, 27:950 (Sept.) 1944.

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The American Academy of Optometry and the publishers of this journal have no objection to the reprinting by other magazines of any of the articles in this issue, provided such reprints are properly credited to the AMERICAN JOURNAL OF OPTOMETRY AND ARCHIVES OF AMERICAN ACADEMY OF OPTOMETRY.

OPTOMETRIC PRACTICE IN THE RURAL AREAS

A current issue of one of our State optometric publications devotes a major part of its editorial space to a discussion of the choice of location for the future practice of the optometric graduate of today.

The discussion did not revolve around the subject of professional versus commercial practice, as the authors took for granted that the present-day graduate would practice professionally—an assumption substantiated by the results of a questionnaire which had been filled in by a large number of graduates showing they all favored professional office quarters and practice.

Instead, the subject discussed was the advantage of locating a practice in a metropolitan area versus a rural community. Both of these

experienced authors[1, 2] strongly favored the idea of the new graduate opening his office in the smaller town. The advantages claimed for this were (1) living costs are lower; (2) office expense is by comparison less; (3) there are greater opportunities to become acquainted quickly with local people; (4) that during the first few years a practice grows faster in the small town than in the large; (5) much less competition, and (6) more good openings.

It was also pointed out that today the larger concentration of optometrists is in the metropolitan centers and for this reason the newcomer has a harder time getting established in a large city. The authors fail to mention that in very great part ophthalmologists also are located in the larger cities, thus adding to the number of professionals doing refractions in these centers.

Certainly a very good case can be presented for the establishment of new practices in the smaller towns. In this connection, the Academy itself is an illustration, as about one-half of its membership practice in small cities and towns, this being a group of men whose practices must meet educational and professional qualifications and of optometrists who must be practicing successfully.

Constituted as we are today in the United States, the small towns offer the new graduate an excellent opportunity to establish himself in professional practice. It is hoped that a large number of these men will avail themselves of this opportunity as they leave college to begin their optometric careers.

CAREL C. KOCH.

SEASON'S GREETINGS

As we go to press, the Academy is in session at Chicago, and the Christmas season is near at hand. The year 1947 is drawing to a close and thoughts turn to the successes of the past and the splendid prospects for the future. In common with optometrists everywhere we are thankful for the science of modern refraction and the skills that are ours which enable us to so properly take care of the visual problems of our patients. On behalf of the Academy we of the Journal staff wish to extend to our subscribers from coast to coast the season's best—a very merry Christmas and a happy, prosperous New Year.

CAREL C. KOCH.

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1. Harry E. Pine. Where Shall I Start My Practice. The Illinois Optometrist. 5.5.51. 1947.
2. Leo Manas. Erst Kommt Das Essen: Dann Die Moral. The Illinois Optometrist. 5.5.53. 1947.

BOOK NOTICES AND REVIEWS

Books received are acknowledged by notices in this department. Special selections will be made for more extensive review in the interest of our readers as space permits.

DISEASES OF CHILDREN'S EYES. James Hamilton Doggart, M.D. Published by the C. V. Mosby Company, St. Louis, Missouri. 288 pages. Illustrated. Cloth. $10. 1947.

The author is an English ophthalmologist who limits his practice to the ocular problems of children. The book is a review of his work. It is a welcome addition to ophthalmic literature, as it presents a review of the subject from the viewpoint of the work done on the infant and small child.

Obviously, when working on young children the history is derived second-hand, no subjective tests can be made and the objective tests which are attempted must be very simple. In addition, the examiner must gain the confidence of the little patient. Doggart properly points out that this is no place for the beginner, (p. 1), as all the diagnostic skill of an experienced hand will be called into play in examining and treating children. He says further, (p. 138), that it is wrong for novices to be entrusted with the prescribing of children's spectacles. This principle needs, he claims, to be emphasized, because many people suppose that a three-months' training in refraction will enable them completely to take charge of a school clinic.

He points out further that headaches due to refractive errors in children are rare, (p. 140); that in ordinary cases free from strabismus it is not necessary to atropise the eyes of children in refraction, (p. 141), and that in myopia cases he believes there is nothing to be gained by under-correcting the refractive error, (p. 145). He also believes that average amounts of close work will not bring on myopia, (p. 145), claiming rather that this visual problem is for the most part hereditary, (p. 144).

In his work he has found that in children sufficiently old to cooperate he secures an adequate view of the fundus in a large percentage of cases without the aid of a mydriatic, (p. 259).

In studying the crystalline lenses of children the author concludes that 90 per cent of all children display some form of cataract (if the term is to include all lens opacities) but he adds, in the majority of cases the cataract is of no serious import as most eyes have harmless congenital lens opacities, (p. 74 and p. 241).

Doggart also observes that styes and blepharitis in children are not caused, as a rule at least, by uncorrected refractive errors, (p. 205 and p. 206).

The author covers the anatomy and developmental processes of the eyes and orbits. He reviews the corrective measures employed, giving considerable credit to orthoptics, (p. 157 and p. 175), in squint cases. The book reviews the diagnostic and treatment techniques used in cases of ocular pathology and also presents the ocular complications and sequels of disease elsewhere.

CAREL C. KOCH.

GIFFORD'S OCULAR THERAPEUTICS. Derrick Vail, M.D. Published by Lea & Febiger, Washington Square, Philadelphia, Pennsylvania. 336 pages. Illustrated. Cloth. $5. 1947.

Prior to Sanford R. Gifford's death in 1944 his Handbook of Ocular Therapeutics enjoyed a reputation for thoroughness unsurpassed by any other book dealing with this subject. The fourth edition, now completely revised, is brought out by Derrick Vail, a professor of ophthalmology at Northwestern University, and a former colleague of Gifford's.

This book deals with the modern non-surgical therapies employed in the treatment of ocular disease. A review indicates that many of these treatment techniques are not too well grounded in either sound clinical procedures, acceptable case reports or in scientific fact and Vail is the first to point out these shortcomings.

Notwithstanding this, he does review the current literature and presents the more carefully made studies of others along with his own extensive background in the field of treating ocular pathology and visual disorders. For instance, he points out that recent claims that progressive myopia is affected by viosterol seem to be without satisfactory evidence, (p 70), and that there is little to support the claims that abnormalities in nutrition, metabolism or the function of the ductless glands are important in the causation of cataract, (p 243). He claims, too, that the widespread exploitation regarding vitamin deficiency in visual problems is obviously extravagant, (p 63).

On the other hand, he finds thiamine chloride to be of some value in treating toxic amblyopias in alcoholics, (p 66); that sulfanilamide is of value in treating trachoma, (p 81); that penicillin, tyrothricin and streptomycin are antibiotics of greatest promise in cases of infections, (p 88). He reports also that sulfonamides and penicillin have reduced

the use of vaccines in the treatment of recurrent hordeola and to some extent in the treatment of blepharitis, (p 109).

The cause of retinitis pigmentosa is, he claims, unknown but may best be considered hereditary. He reviews many suggested therapies but finds little evidence to support any, (p 317-318).

In discussing the use of cycloplegics Vail is far from charitable. While Vail admits the widespread use of cycloplegics among ophthalmologists he urges that these drugs be used with caution. He says, (p 52):

"Atropine *should never be used for ophthalmoscopic examination* except in infants whose pupils do not react to weaker derivatives. In using atropine several precautions must be observed. It must be remembered that 1 minim of 1 per cent solution contains 1/100 grain of atropine sulphate, and that a large part of this finds its way by the nasolacrimal duct into the nasopharynx, where it is absorbed into the circulation. *Hence in babies it must be used with caution*, as fatalities have been observed following absorption of atropine in this way. . . . Since atropine causes a rise of intraocular tension in cases of glaucoma *one should be sure that the tension is not elevated before it is given . . .*"

The italics are Vail's.

Regarding homatropine he finds this the drug most employed in ophthalmological refraction, despite the knowledge that the resultant cycloplegia is not complete. Regarding the dangers of its use, he says, (p 53):

"The same dangers are involved in the use of homatropine as with atropine, but these may be more easily counteracted or avoided. Acute attacks of glaucoma have been brought on after using homatropine in refraction. This is especially to be feared in persons above the age of forty years, but may also occur in younger patients. Although there must exist a predisposition to glaucoma in such cases, it is impossible to diagnose such a predisposition beforehand, and the *only safe procedure when using homatropine in refraction is the use of a miotic immediately afterwards* for all patients except children."

The italics are again Vail's.

In discussing myopia, (p 320), Vail claims that the scientific observer will approach attempts at therapy with a certain skepticism because of what is known as to the pathogenesis of the condition. He suggests the use of glasses and advice on personal hygiene in these cases and makes no mention of orthoptics.

In the chapter on physical therapy Vail reviews the work of Duke-Elder, Vogt and others as this relates to the effect of infra-red rays on the crystalline lens and the author points out that nearly all ultra-violet rays are absorbed by the cornea and do not enter the inner portions of the eye in appreciable amount, (p 130-131).

In the section dealing with drugs used for instillation the author discusses at some length the pH factor, which will be of interest to those optometrists who prescribe contact lenses, (p 55-56).

This is a well put together ophthalmological text book. It is readable and the authors, first Gifford and then Vail, present theories and factual material clearly. It is of value to optometrists as it reviews the newer techniques used by physicians in treating ocular disease and presents the conservative thinking of ophthalmology towards this portion of their work.

CAREL C. KOCH

TRANSACTIONS OF THE AMERICAN ACADEMY OF OPTOMETRY

A department devoted to announcements, reports, appointments, organization data, news, professional problems and ideals, as these relate to the Academy.

A full report of the annual meeting of the American Academy of Optometry which has just been concluded in Chicago will appear in the January 1948 issue of the Journal.

CHAPTER ACTIVITIES

MINNESOTA CHAPTER

Dr. Walter H. Nordin, Austin, was elected 1948 president of the Minnesota Chapter, American Academy of Optometry, at its winter meeting held November 9, 1947. Other officers elected to serve the coming year were Dr. Carel C. Koch, Minneapolis, vice-president, and Dr. Rudolph H. Ehrenberg, Granite Falls, secretary-treasurer. Drs. E. W. Larson, Hibbing; A. G. Engel, Little Falls; J. W. Shimek, Minneapolis, and Robert P. Best, Fairmont, were elected to the executive council.

The first meeting for 1948 was scheduled for January 25, 1948, and tentative plans made for engaging a speaker from the psychiatry department, University of Minnesota.

SOUTHERN CALIFORNIA

Dr. James Gregg, a faculty member of the Los Angeles School of Optometry, was the speaker at the October 10th meeting of the Southern California chapter. Dr. Gregg reviewed Dr. Meredith Morgan's paper which appeared in the September issue of the Journal, "Stereoscopic Targets in Orthoptics"—a report of an investigation financed in part by the American Academy of Optometry.

During the second half of the meeting members were shown slides on ocular pathology accompanied by a lecture by William L. Cooper, M.D., pathologist of the Cooper Pathological Laboratories.

The following were elected to office for 1948: Dr. John R. Dean,

president: Dr. Robert Graham, vice-president, and Dr. E. Clarke Deacon, secretary-treasurer.

MARYLAND CHAPTER

The Maryland chapter held a dinner meeting October 24, 1947 at the Stafford Hotel, Baltimore, at which Dr. Louis Hill lectured on "Professional Economy."

Accepted into membership was Dr. J. Lawrence Ebauer.

CURRENT COMMENTS

Virginia Huck
Editorial Assistant

Optometrists will confer a favor by sending news items of general interest for this department, such as relate to new instruments, clinical techniques, education, visual health and optometric legislation and organization.

K. CLIFFORD HALL JOURNAL VISITOR

The Journal's third British visitor this year brought first hand accounts of recent experiences of austerity-pinched Britons and the picture was not a pleasant one.

Each person is allowed one egg a month, an ounce or two of meat each week; potatoes are ample, but rationed; coal is scarce. These are a few of the facts as related to the Editor by Mr. K. Clifford Hall, British optometrist who toured the United States in November. A specialist in contact lenses—and a pioneer in the field—he came to the United States to study our methods of manufacture and fitting.

A brighter side of the picture was Mr. Hall's account of the London Refraction Hospital's jubilee congress which he attended in October.

The London Refraction Hospital, a non-medical clinic staffed entirely by optometrists, celebrated its 25th anniversary at a four-day meeting which was attended by a host of Britain's outstanding leaders in optometry. Mr. Hall reported that many excellent papers on optometric problems were presented at the meeting.

A GOOD PUBLIC RELATIONS YEAR

During 1947, hundreds of press releases containing news of optometry and related fields came into the Journal office—the best barometer possible that optometrists everywhere are paying more and more needed attention to public relations.

This department would have liked to run all the items. Obviously, space would not permit this, but in the past twelve months we have sifted out and passed on to our readers what we believed to be the significant activities in optometry. We shall continue to do this in 1948—with the continued help of those who have kept us posted in 1947.

But 1947 is not yet over, and the last few weeks have been full of optometric activities everywhere.

INDIANA "U" TO ESTABLISH OPTOMETRY SCHOOL

Indiana University officials announced the possible adding of a course in optometry to the university curriculum. This information was sent to the Journal by Dr. R. W. Tubesing, Academy Fellow and member of the Indiana Optometric Association. We hope further details of this important news will develop soon.

FIFTH NEW JERSEY INDUSTRIAL CONFERENCE HELD

New Jersey optometrists heard two more lectures on occupational optometry on December 10. These lectures are co-sponsored by the New Jersey Chapter, American Academy of Optometry, and the New Jersey Optometric Association. The December meeting was the fifth to date. Six more have been scheduled.

NEW ENGLAND OFFICERS DISCUSS OPTOMETRY'S FUTURE

State officers of six state optometric societies, recently organized into the "New England Officers' Conference," held their second meeting to interchange ideas for the development and progress of optometry in New England. The meeting was held at the Hotel Statler, Boston, on November 2.

BANNON TO SPEAK AT INDIANA CONVENTION

Dr. Robert E. Bannon, Fellow, American Academy of Optometry, and faculty member, Columbia University School of Optometry, will discuss "Evolution and Essentials of Refraction," at the 51st annual convention of the Indiana Optometric Association.

The convention is scheduled for January 18, 19, and 20, 1948, at the Severin Hotel, Indianapolis.

TEMPLE UNIVERSITY READING CLINIC DATE CHANGED

Temple University, Philadelphia, has announced the following change in schedule for its 1948 Mid-winter Institute on Reading Disabilities. Originally scheduled for January, the date has now been advanced to February 2 to February 6 inclusive.

VOLUME 24, 1947, GENERAL INDEX

This is an index covering all of the departments of the AMERICAN JOURNAL OF OPTOMETRY AND ARCHIVES OF AMERICAN ACADEMY OF OPTOMETRY. The letters used to explain in which department the material indexed appears are as follows: (OP), Original paper; (E), Editorial; (T), Transactions of the Academy; (CC), Current Comments by Virginia Huck; (SR), Special Reports; (C), Correspondence; (Ab), Abstracts; (BN), Book Notices, and (A), Announcements.

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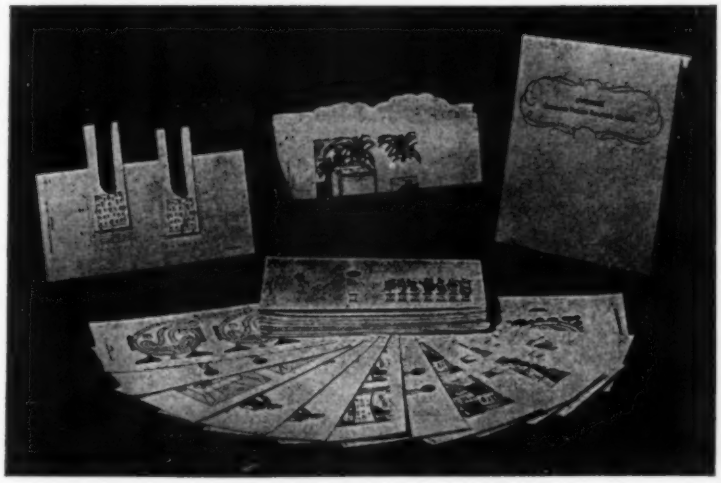

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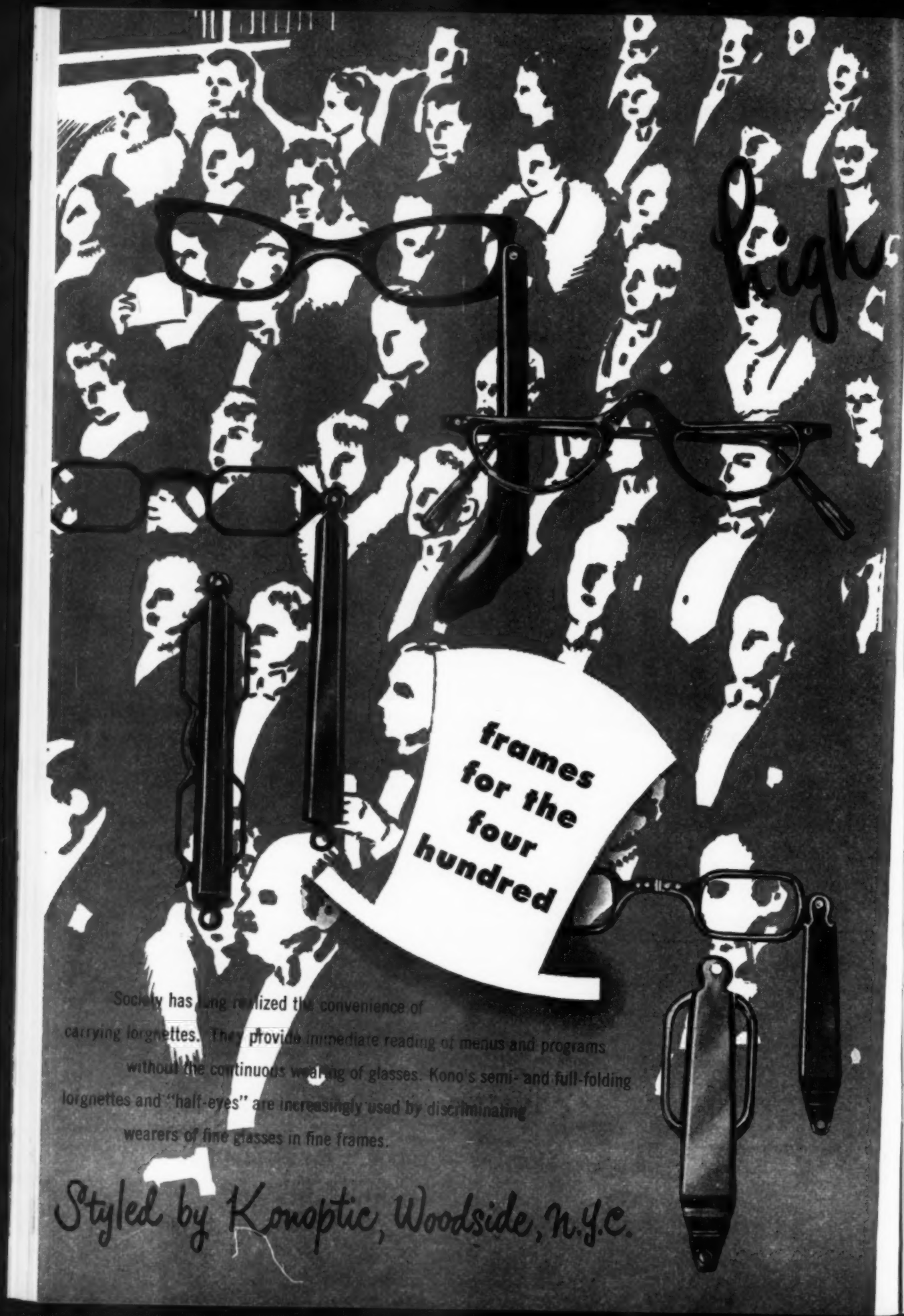
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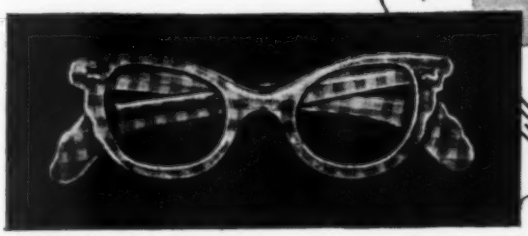

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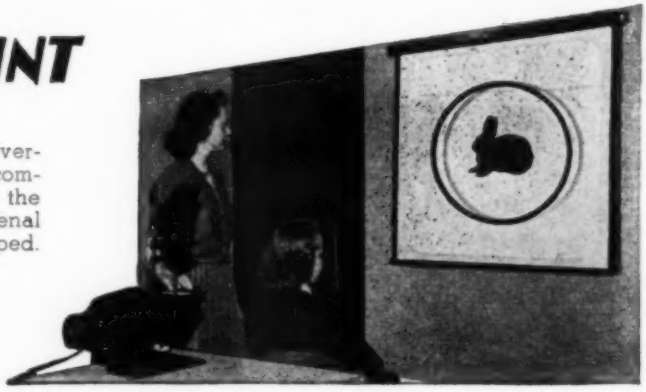

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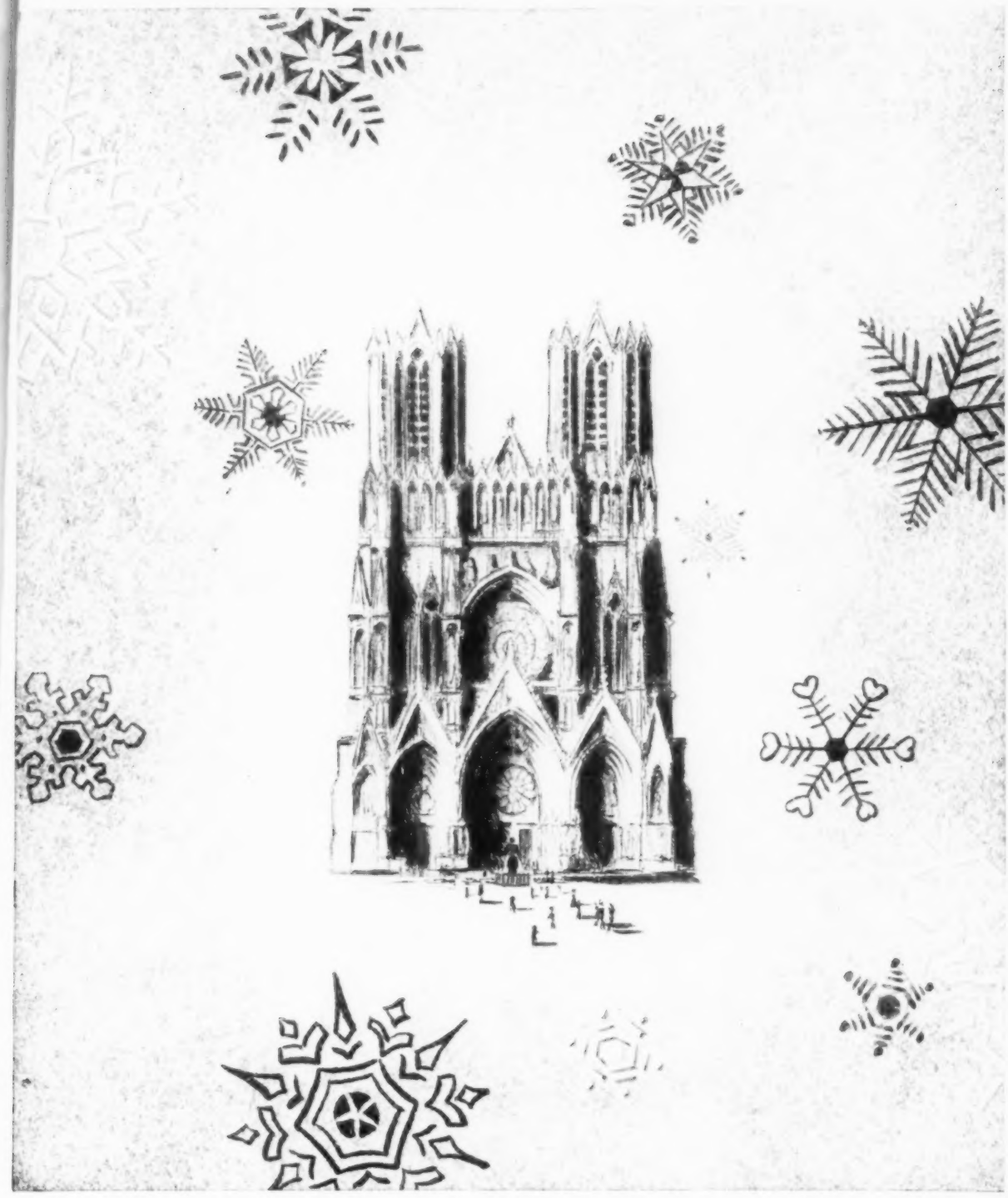

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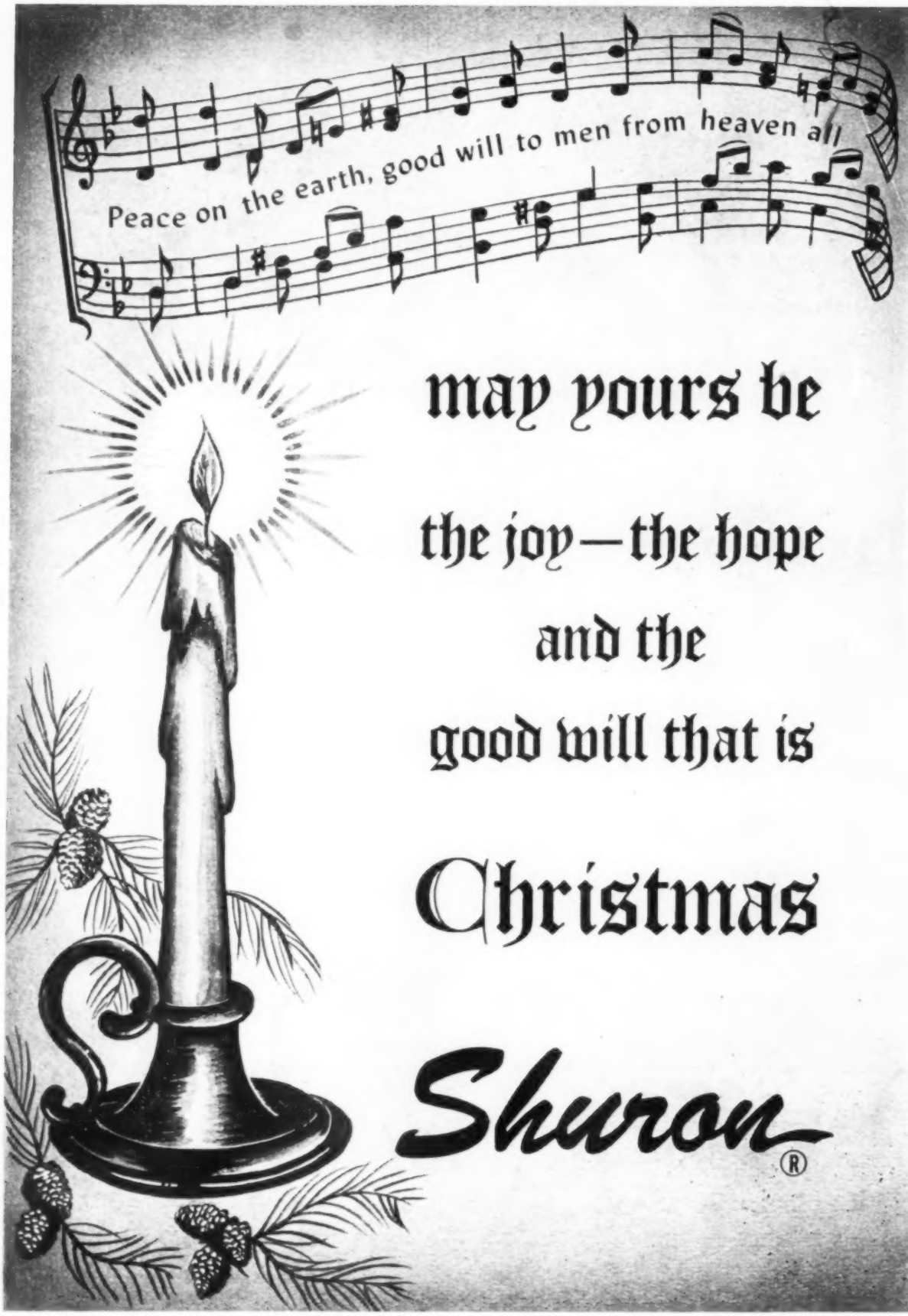
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